

ALTERED IA SENSORY FIBER MEDIATED RESPONSES IN
INDIVIDUALS WHO HAVE HAD POLIO

By

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Dedicated to the Women and Men Who have had
Polio

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Ia sensory fiber mediated responses of joint position sense and H-reflex modulation were studied in persons with and without a history of polio. The joint positioning sense experiment consisted of a slow velocity passive knee flexion task with and without vibratory perturbation. Analysis of the joint positioning accuracy means revealed a significant Group x Vibratory Condition interaction for constant error and absolute error. Significant main effects for the vibration condition were also found for absolute constant error.

H-reflex experiments were conducted to determine Hmaximum/Mmaximum ratios, level of inhibition resulting from low frequency electrical stimulation, level of inhibition resulting from tendon vibration, and level of facilitation following a low level voluntary

muscle contraction. Analysis of the Hmaximum/Mmaximum ratios indicated a significant group effect; the polio group demonstrated a reduced Hmaximum/Mmaximum ratio in comparison to the non-polio group. The other H-reflex experiments, though showing a trend in group differences, failed to reach statistical significance. These findings are interpreted as evidence supporting the hypothesis of altered Ia sensory mediated functions in individuals who have had polio.

CHAPTER 1 INTRODUCTION

Sensorimotor integration, or two-way communication between the motor and sensory systems, is a theory which describes an essential and fundamental aspect of the organization and functioning of the human neuromuscular system (Brooks, 1986; Glencross, 1995). This integration of sensory and motor functions is theorized to occur across and throughout the length of the neural hierarchy (MacKenzie & Marteniuk, 1985; Marteniuk & MacKenzie, 1980). The nature of sensorimotor integration is such that the motor and sensory systems are mutually dependent. Consequently, dysfunction of the sensory system may lead to deficits in motor function, and likewise dysfunction of the motor system may lead to deficits in sensory function (Duncan & Badke, 1987; Monster, Herman, & Altland, 1973). Ample evidence demonstrates the impact that peripheral or central sensory dysfunction has on impairing motor function (Haaland, Harrington, & Yeo, 1987; Roy, 1978; Sainburg, Poizner, & Ghez, 1993; Teasdale et al., 1994). However, the evidence for sensory sequelae as a result of primary motor dysfunction is less well-developed. That is, primary motor pathology without concomitant sensory pathology is rare. Therefore, the purpose of this dissertation was to present polio as a model of primary motor disease, tested within the domain of sensorimotor integration. Specifically this series of experiments investigated the sensory functions mediated by the largest afferent sensory fibers (type Ia) from the muscle spindle.

Background

Acute poliomyelitis is a viral disease that primarily affects the motor neurons of the CNS, particularly the gray matter of the anterior spinal cord (Leestma, 1991). The late effects of polio, occurring 25 or more years after the acute disease, are also attributed to motor neuron dysfunction (Agre & Rodriguez, 1990; Dalakas, 1995b). These chronic effects of polio, currently termed postpolio syndrome (PPS), include increased muscle weakness, easy fatigability, muscle pain, and joint pain (Agre & Rodriguez, 1990; Campbell, 1993; Halstead, 1995). There are an estimated 1.5 million people in the United States who have had polio, and though the exact incidence of PPS is not known, researchers predict that 25% to 100% of all individuals who have had polio will develop PPS (Birk, 1993; Brummel-Smith, 1993; Bruno, Cohen, Galski, & Frick, 1994; Stone, 1994).

The underlying pathophysiologic changes that are known to contribute to the late effects of polio include myoneural junction dysfunction, a reduction in available motor units, overuse weakness, disuse atrophy, and alterations in motor neuron excitability (Agre, Rodriguez, & Tafel, 1991; Melchers et al., 1992; Ravits, Hallett, Baker, Nilsson, & Dalakas, 1990; Wiechers, 1995). Additionally, magnetic resonance imaging has furthered an understanding of postpolio pathology by identifying lesions in the cerebral cortex, thalamic-cortico pathways, and the reticular activating system in individuals who have had polio (Bruno, Frick, & Cohen, 1991; Bruno, Cohen, Galski, & Frick, 1994). Though lesions outside of the voluntary primary motor pathway are now recognized as possibly contributing to PPS, early pathological studies following acute polio clearly demonstrated that polio can involve neural components of the sensory system. In particular, lesions of

the dorsal root ganglion, intermediate horn of the spinal cord, vestibular nuclei, and reticular activating system were common in severe cases of polio (Bodian, 1949).

The potential for degraded sensory function as a late effect of polio is an area that has received little attention. Current research has focused on the dominant role of motor neuron damage. However, patient reports of balance difficulties, magnetic resonance imaging findings, and altered motor neuron responses to reflex testing suggest that specific sensory functions may be involved (Agre & Rodriguez, 1990; Bruno, Frick, & Cohen, 1991; Soliven & Maselli, 1992). These clinical findings, coupled with sensorimotor integration predictions of altered sensory function in response to motor dysfunction, provided the basis for questioning the claim of normal sensation in individuals who have had polio. Specifically, the sensory Ia fibers with direct connections to both the muscle spindle and motor neurons are likely candidates to demonstrate functional changes. Converging lines of theoretical evidence that supported the possible alteration of Ia sensory mediated functions are discussed next.

Sensorimotor integration provides the theoretical underpinnings that supports the notion of a deficit in the functions mediated by Ia sensory nerve fibers in individuals who have had polio. The keystone of sensorimotor integration is that motor function and sensory perception are fundamentally and inexorably linked (Glencross, 1995; Marteniuk & MacKenzie, 1980). This linkage is of such a form that sensation shapes motor function, and conversely motor function shapes sensation (MacKenzie & Marteniuk, 1985; Marteniuk, 1992). The unique anatomical and physiological arrangement of Ia sensory nerve fibers to intrafusal muscle fibers and motor neurons provides a clear testing ground

for the utility of sensorimotor integration as operating at the spinal level (Gordon & Ghez, 1991).

The most likely sensory dysfunctions in the presence of a primarily motor lesion, such as that seen in postpolio, are elements of proprioception and reflex modulation mediated by the Ia sensory nerve fibers. While this statement is made from the theoretical stance of sensorimotor integration, it is also supported by physiological findings which clearly illustrate structural relationships supporting the strength of sensorimotor relationships, particularly between anterior horn cells and Ia sensory fibers (Brooks, 1986). A strong finding, not specific to polio, is the effect of neuron death on the health of closely associated nerve cells. The death of a neuron can have profound, and even fatal consequences for nerve cells that have strong synaptic contacts with the dead or diseased nerve cell (Jessel, 1991). In the case of polio where motor nerve cell death or severe injury is common, the strong monosynaptic pathways that connect the Ia nerve cell to the motor cell would increase the possibility of subsequent Ia sensory nerve cell death or damage (Allen et al., 1994; Soliven & Maselli, 1992).

An additional example of how physiology corroborates sensorimotor integration concerns the connection of the Ia sensory endings with the intrafusal muscle fibers. The Ia sensory ending responds to stretching of the intrafusal muscle fiber. The intrafusal muscle fiber's sensitivity to stretch is under dynamic supraspinal control. These supraspinal influences are in part mediated by sensory Ia fiber stimulation. Thus, Ia sensory information is related to motor drive, and motor drive is mediated by Ia sensory input (Brooks, 1986). Consequently, a deficit in either motor drive or intrafusal muscle function has the potential to alter Ia mediated sensory function.

While the previous discussion provides a strong basis for suggesting that Ia mediated sensory functions are altered based on sensorimotor integration and neurophysiological correlates, the pathophysiology of polio may also contribute to the degree of sensory involvement. The commonly accepted portal of entry for the polio virus into the CNS is via the blood stream, and researchers have determined that one site of entry for the polio virus is through the myoneural junction (Illa, Leon-Monzon, Agboatwalla, Dure-Samin, & Dalakas, 1995; Leon-Monzon, Illa, & Dalakas, 1995). Furthermore, there is a propensity for the polio virus to more easily invade larger neurons (Leestma, 1991). Bodian (1949) has reported lesions in the dorsal root as a result of polio infection, though the type of nerve involved was not specified. If the same propensity for size holds true for polio to enter sensory neurons and if a portal of entry is through the muscle nerve connection, the Ia sensory nerve fibers, being among the largest of peripheral nerve fibers and having close connections to intrafusal muscle fibers, may be a target during the acute infection.

Several interacting factors have the potential to alter Ia sensory nerve mediated functions in polio. Sensorimotor integration tenants indicate that a sensory alteration should be present as a result of severe motor disease. Therefore, experiments specifically designed to test those functions mediated by the sensory Ia nerve fibers may provide findings of altered sensory functions in what is believed to be a primary motor pathology. The functions mediated by the sensory Ia fibers include joint position sense and motor reflex modulation, and these will be discussed next.

Ia sensory nerve fibers are stimulated *in vivo* by intrafusal muscle fiber stretch or changes in rate of stretch (Brooks, 1986; Guyton, 1991). Physiologically the stretch

related information is used for joint position sense (one aspect of proprioception) and for modulation of spinally mediated reflex arcs (Desmedt, 1983; Matthews, 1990). Based on these two distinct roles of the Ia sensory nerve fiber, experimental paradigms that selectively manipulate the presentation of Ia sensory nerve fiber information may be used to test for changes of Ia nerve fiber function in individuals who have had polio. Specifically, manipulating Ia nerve fiber function with vibratory and electrical stimulation in a systematic manner can help to discern changes in Ia function in joint position sense and as a key component in reflex modulation (Desmedt, 1983, Matthews, 1990).

The role of the Ia sensory nerve input in joint position sense has been the focus of many investigations (Capaday & Cooke, 1983; Redon, Hay, & Velay, 1991; Roll & Vedel, 1982). The key finding of these studies has been that Ia sensory input is of considerable importance (Capaday & Cooke, 1981; Roll, Vedel, & Ribot, 1989). In particular, the Ia sensory input from the lengthening muscle, whether relaxed or eccentrically contracting, provides valuable input contributing to the accuracy of joint position perception (Inglis & Frank, 1990; Inglis, Frank, & Inglis, 1991; Redon, Hay, & Velay, 1991). The experimental paradigm that has been frequently used in studying Ia mediated joint position sense involves the use of low amplitude vibratory perturbation at a frequency of 80-100 Hz. This level of vibration when applied directly over a muscle tendon strongly stimulates the Ia nerve fibers (Cordo, Gandevia, Hales, Burke, & Laird, 1993). In response to this type of vibratory stimulation individuals experience a kinesthetic illusionary movement if the limb is stationary, or experience an inaccuracy (undershooting) during a dynamic limb positioning task (Inglis & Frank, 1990; Inglis, Frank, & Inglis, 1991). Individuals who have had polio may show an altered response to

vibratory perturbation of disease weakened muscles. If the Ia sensory nerve fibers are not functioning to the same degree as observed in normal individuals, then a change in the vibratory effect on joint positioning error is predictable.

The Ia mediated muscle stretch reflex and the electrically related Hoffmann (H) reflex have been subjected to extensive research (Burke, 1983; Hugon, 1973; Matthews, 1986; Schieppati, 1987; Taborikova & Sax, 1968). The simplest description of the muscle stretch or myotactic reflex is that it represents a two neuron arc of the Ia sensory nerve fiber and the alpha motor nerve fiber (Brooks, 1986; Guyton, 1991). Consequently, a lesion of either the motor pathway or sensory pathway of the arc will result in an abnormality of the evoked reflex (Kimura, 1989). The H-reflex is the electrical analogue to the muscle stretch reflex, generated by an electrical impulse tailored to selectively stimulate Ia fibers (Fisher, 1992).

In reality, both the mechanically generated stretch reflex and the electrically generated H-reflex have monosynaptic and polysynaptic components, and are influenced by multiple factors such as other motor and sensory segmental inputs along with descending supraspinal inputs (Burke, 1983; Hugon, 1973; Kameyama, Hayes, & Wolfe, 1989). However, well-established experimental designs have been developed to determine the segmental functional relationships between Ia sensory nerves, interneuronal pools, and motor neuron pools. Depending on the experimental set-up, Ia sensory information can have excitatory or inhibitory influences on specified homonymous (same) or heteronymous (different) motor neuron pools (Cordo, Gandevia, Hales, Burke, & Laird, 1993; Hultborn, Meunier, Morin, & Pierrot-Deseilligny, 1987; Hultborn, Meunier, Pierrot-Deseilligny, & Shindo, 1987; Van Boxtel, 1986). Also, alterations in stimulation conditions such as

stimulation during vibration, or immediately following volitional contraction, or using higher stimulation frequencies all have predictable effects mediated by Ia fibers (Bongiovanni, Hagbarth, & Stjernberg, 1990; Cook, 1968; De Gail, Lance, & Neilson, 1966; Desmedt, & Godaux, 1978; Van Boxtel, 1986). Thus, by selectively manipulating the conditions just prior to or during electrical stimulation, the H-reflex can be used to determine the functional status of Ia sensory nerves as they mediate motor reflex arcs.

Closely aligned with the discussion of the muscle stretch reflex is the tonic vibration reflex (TVR). The TVR is a slowly building tonic muscle contraction in response to the mechanical vibration, which stimulates the Ia sensory fibers (Desmedt & Godaux, 1978). This response is polysynaptic and requires facilitory influences from higher centers (Desmedt, 1983). Because of the direct relationship between vibratory stimulation of Ia fibers and the TVR, the TVR is also potentially useful in determining the functional status of the Ia system.

In summary, by approaching the question of Ia function in postpolio individuals from two distinct perspectives, joint position sense and reflex modulation, a global picture may be provided of Ia mediated function. First, an experiment that tested joint position sense and its perturbation by vibration is used as a mechanism to elucidate the Ia nerve fiber's function in mediating joint position sense through the sensory system to perception. Second, a series of H-reflex and TVR experiments test the Ia sensory fiber's contribution to spinal segmental reflex function. Taken together, the proposed series of experiments provide converging lines of evidence concerning the functions mediated by Ia sensory nerve fibers in individuals who have had polio.

Statement of the Problem

Clinically, individuals who have had polio are thought to be without sensory involvement. However, based on the tightly coupled relationship between sensorimotor systems, a deficit in sensory function is expected in the presence of severe motor pathology. Thus, the purpose of this study was to critically examine those functions, joint position sense, and muscle reflex modulation mediated by sensory Ia nerve fibers in individuals who have had polio.

Hypotheses

Based on sensorimotor integration and polio as a model of primary motor pathology, seven testable hypotheses were generated. The hypotheses are grouped into two areas, joint position sense and reflex modulation.

Joint Position Sense Hypotheses

When compared with age- matched control participants, participants who have had polio will

- (1) Demonstrate significantly greater error in a knee joint flexion position matching task; and
- (2) Demonstrate significantly less error in a knee joint flexion position matching task perturbed by vibration.

These hypotheses were developed within the context of sensorimotor integration theory (Glencross, 1995; MacKenzie & Marteniuk, 1985; Marteniuk & MacKenzie, 1980). Given that propositions of sensorimotor integration theory include; (1) sensory deficits may cause motor dysfunction, and (2) motor deficits may cause sensory dysfunction, a sensory deficit should accompany a primary motor disease. Because of the

closely aligned nature of the Ia sensory nerve fibers connections with motor structures damaged by the polio virus, the Ia sensory mediated responses were thought to be likely indicators of sensory involvement. Furthermore, these hypotheses concerning altered joint position are justified based on the findings that document a strong role of the Ia sensory fibers in mediating joint position sense and the effects of vibratory perturbation on altering joint position sense (McCloskey, 1973; McCloskey, Cross, Honner, & Potter, 1983, Inglis & Franks, 1990).

Reflex Modulation Hypotheses

When compared with age-matched control participants, participants who have had polio will show a significant reduction in

- (1) Hmaximum /Mmaximum ratios;
- (2) voluntary contraction facilitation;
- (3) vibratory inhibition;
- (4) low frequency electrical inhibition; and
- (5) the TVR.

These reflex modulation hypotheses were also developed within the context of sensorimotor integration theory (Glencross, 1995; MacKenzie & Marteniuk, 1985; Marteniuk & MacKenzie, 1980). Specific to the reflex modulation functions of the Ia sensory system, sensorimotor integration theory predictions of altered sensory function in the presence of a motor disease may take the form of changes in motor reflex modulation in individuals who have had polio. The examination of H-reflexes as a research method has a long history of use in discerning neurological function and changes with pathology of central and peripheral origins (Fisher, 1992; Goodgold & Eberstein, 1984; Kimura, 1989).

Specific to polio, altered H-reflex responses have been previously reported in the literature (Prakash, Sinha, Mukherjee, & Dey, 1995; Soliven & Maselli, 1992). With the selective manipulation of stimulation characteristics and patterns, Ia sensory fiber mediated function can be delineated from the motor contribution of the reflex arc (Cook, 1968; Desmedt & Godaux, 1978; Hultborn, Meunier, Pierrot-Deseilligny, & Shindo, 1987; Matthews, 1990; Van Boxtel, 1986). Specific to the TVR, if a differential pattern is noted between the H-reflex responses and the TVR, additional insight may be gained concerning the involvement of monosynaptic versus polysynaptic Ia pathways (De Gail, Lance, & Neilson, 1966)

These joint position sense and reflex modulation hypotheses are based on strong theoretical grounds, pathophysiologic findings, and reports in the literature. The overall question of Ia sensory fiber dysfunction in individuals who have had polio is supported by the fact that very few researchers have critically examined systems other than the lower motor neuron in persons who have had polio. In part, this apparent lack of interest in other possible contributing factors in postpolio is related to the severity and preponderance of primarily motor deficits. Additionally, individuals who have had polio generally demonstrate adequate motor control, whereas cutaneous sensory deficits are often explained by peripheral nerve or spinal nerve entrapment syndromes (Perry, Barnes, & Gronley, 1988; Windebank, 1995). However, systematic testing of Ia sensory fibers has not been conducted.

Several investigators concerned with acute and postpolio individuals have acknowledged the possibility of sensory pathology in those sensory systems related to motor control (Bodian, 1949; Solivan & Maselli, 1992). One reason Ia mediated sensory

dysfunctions may not be overtly apparent is that they are overshadowed by the primary muscle weakness. Furthermore, because polio frequently results in incomplete paralysis, enough multijoint function remains to maintain coordinated movement. Isolated testing of the sensory Ia fibers as they function specifically in joint position sense and as reflex modulators of a monoarticular muscle can provide information concerning the late effects of polio on the function of sensory Ia fibers.

Limitations

There are three primary limitations of this dissertation; (a) the specifics of polio pathology, (b) inclusion criteria for those individuals who have had polio, and (c) experimental methodologies used in examining Ia sensory mediated functions. As this study uses polio as a model of a primary motor disease, the findings are generalizable only to polio. Additionally, it is known that though polio is primarily a disease of the anterior horn cell, other areas of the central nervous system can be involved. Even more specifically, inclusion criteria for polio participants is based on the degree of quadriceps weakness; participants with polio will be selected who have good to poor strength of the quadriceps muscle. Thus, these findings may not be generalizable to postpolio individuals who have normal, trace, or no function of the quadriceps muscle.

The planned experimental methodologies also serve as a source of limitation. Vibratory frequency and amplitude are controlled to fairly tight specifications. The constraints placed on the vibration characteristics may decrease the effectiveness of vibration in stimulating the Ia sensory fibers, resulting in smaller effect sizes. Conversely, vibration while known to be strongly specific for Ia sensory fiber stimulation can also

stimulate other sensory nerve fibers. Thus, some of the vibratory effects may represent stimulation of additional sensory nerve fiber types.

Concerning the H-reflex studies a primary limitation is that altered H-reflex responses are not necessarily only attributable to dysfunction of the Ia sensory system. Furthermore, H-reflex responses are highly variable as is the level of dysfunction within the polio group. Lastly, the quadriceps is used as the target for vibratory perturbation of joint position sense and the reflex modulation studies. The findings of this dissertation are specific to the quadriceps, and generalization to other muscles may not be appropriate.

Significance

The findings from this series of experiments may advance our knowledge concerning the pathophysiology of late polio, aid in the development of rehabilitation programs for primary motor diseases, and contribute to the elaboration of sensorimotor integration theory. This dissertation presents polio as a model by which the concepts of sensorimotor integration theory are challenged. Specifically, with polio as a primary motor disease, sensorimotor integration theory predicts that the disease process affecting the motor components should have some negative effect on sensory components. In the case of polio, the sensory system that should show the greatest impact of motor disease is the sensory Ia fibers from the muscle spindle. These fibers are indirectly tested according to their physiological and behavioral functions, as proprioceptors mediating joint position sense and as the afferent arc of the H-reflex and TVR response. Based on the expected results of these experiments four potential points of significance are discussed in the following paragraphs.

First, this dissertation will contribute to the body of literature concerning joint position sense. Several investigations have been conducted on knee joint position sense, addressing issues related to normal function, fatigue effects, and aging effects (Perlau, Frank, Fick, 1995; Skinner, Barrack, & Cook, 1982; Skinner, Wyatt, Hodgdon, Conrad, & Barrack, 1986). This project will further knowledge by studying the effect of vibratory perturbation applied during a dynamic knee joint positioning task. The vast majority of joint position sense studies using vibratory perturbation have been conducted on upper extremity joints or the ankle and great toe joints. The application of this technique may prove to have other roles in future investigations including acute and chronic knee joint injuries or possible effects related to knee surgery and joint replacement.

Secondly, the H-reflex and TVR portion may identify changes occurring in the sensory loop of the evoked reflex. Many investigators have reported that specific manipulations of stimulus type and stimulus site can document reflex modulation directly attributable to monosynaptic and polysynaptic connections of the Ia sensory fibers (Ashby, Stalberg, Winkler, & Hunter, 1987; Burke, Gandevia, McKeon, 1984; Hultborn, Meunier, Morin, & Pierrot-Deseilligny, 1987; Hultborn, Meunier, Pierrot-Deseilligny, & Shindo, 1987; Van Boxtel, 1986). However, this project will extend these reflex modulation experimental techniques to specifically identifying a loss in function of the Ia sensory fibers as a result of polio pathology.

Thirdly, the results may improve the clinical ability to treat individuals who are postpolio. By identifying changes in proprioception and reflex modulation, treatment programs may be developed that recognize and specifically address the functional limitations caused by a dysfunctional sensory component in the sensorimotor system.

Currently treatment for individuals suffering from the late effects of polio focuses on maintaining muscular strength, preventing fatigue, and decreasing pain. If evidence is developed identifying a sensory dysfunction in late polio, the use of other treatment approaches such as balance retraining, coordination exercises, and movement reeducation might be pursued. Furthermore, impetus to reevaluate other motor pathologies for potential sensory deficits may develop from these planned experiments.

Lastly, the findings will contribute to the development and delineation of sensorimotor integration theory. The question of motor deficits resulting in sensory system alterations, while predicted by theory, have not been documented in polio. If the Ia sensory fiber mediated functions are altered then, additional evidence in support of sensorimotor integration theory will be identified.

To reiterate, historically polio is thought to be a primary disease of the motor neuron. The sensorimotor integration theory predicts a reversible role of sensation shaping motor function and motor function shaping sensation. Polio is presented as a model of primary motor disease to be used in testing the role of altered motor function on sensation.

CHAPTER 2

REVIEW OF THE LITERATURE

This literature review is presented in five sections. The first section includes a presentation of sensorimotor integration theory and the neurophysiologic correlates. The second section provides a discussion of the pathophysiology of the acute and chronic effects of polio. The third section discusses the muscle spindle as a motor-sensory organ with vital roles in joint position sense and reflex modulation. The fourth section presents details concerning the role of the muscle spindle and in particular the Ia sensory fibers' contribution to joint position sense. The fifth section focuses on the use of muscle reflex testing as a method of measuring Ia sensory nerve contributions to reflex integrity.

Sensorimotor Integration and Motor Control

Sensorimotor Integration

Sensorimotor integration is a theory that has been a cornerstone of investigations into the neuromuscular control of movement spanning across the disciplines of neuroscience, physical science, and psychology (Gallistel, 1980; Lashley, 1917; Requin, Semjen, Bonnet, 1984; Sherrington, 1906; Whiting, 1984). This complex sensorimotor relationship is often presented in the form of a process, operating throughout the nervous system, inclusive of central and peripheral contributions to the sensorimotor process (Allison, 1995; Burtner & Woollacott, 1995; Dean & Cruse, 1995; MacKenzie & Marteniuk, 1985; Narendra, 1995). Additionally, sensorimotor integration is not an issue of controversy within other theories of motor control which may polarize between

concepts of prescriptive versus emergent aspects of motor behavior (MacKenzie & Marteniuk, 1985; Marteniuk, 1992; Turvey, 1990; Whiting, Vogt, & Vereijken, 1992).

The term sensorimotor integration or the description of sensorimotor interactions is frequently used in the discussion of motor control issues (Abbs, Gracco & Cole, 1984; Conner & Abbs, 1990; Marteniuk & MacKenzie, 1985; Marteniuk, 1992; Massone, 1995). Glencross (1995) specifically states that the term sensorimotor integration and perception-action-perception are both descriptive of the same process; a cyclical process that cannot be understood if the systems are studied in isolation from one another. This recognition of the inseparable coupling between sensorimotor processes is given further credence by being included in the so many other motor control and motor learning theories (Adams, 1971; Arbib, 1995; Frank & Earl, 1990; Mulder, 1991; Proteau, 1992; Schmidt, 1988).

Marteniuk and MacKenzie (1980) describe sensorimotor interaction as the complex process of combining sensory afferent neural inflow with motor efferent neural outflow, throughout the neural axis. Specifically, Marteniuk (1992) states that “the actual expression of coordinated movement, however, is modified by an interplay of motor and sensory information that fine tunes movement to the specific context of the situation.”(p.119). The model of sensorimotor integration presented by Marteniuk and MacKenzie (1980) was developed during studies of interlimb coordination in an effort to account for the interaction effects or crosstalk between simultaneously moving limbs. The core idea expressed in the model involves multiple sites along and across the neural axis where sensory inflow can modify motor outflow and motor outflow can modify sensory inflow. Apparently the impetus for the development of this theory seems to have been a

desire to integrate structural and physiological findings with behavioral science findings as related to coordinated interlimb movement.

In a series of experiments based on this model of sensorimotor integration, Marteniuk, MacKenzie, and Baba (1984) have shown that the errors associated with bimanual movements are predictable and to be expected. Furthermore, sensorimotor integration takes into account the polymodal aspect of sensory information (many different sensory modalities) both exteroceptive and interoceptive, as it relates to proprioception and motor control (Marteniuk, 1992). This concept of exploring sensorimotor integration across different tasks and different sensory modalities continues to be an area of vigorous research activity (Abbs, Gracco, & Cole, 1984; Chicoine, Lassonde, & Proteau, 1992; Proteau, Marteniuk, & Levesque, 1992; Proteau, Charest, & Chaput, 1994; Wolpert, Ghahramani, & Jordan, 1995).

Neurophysiological Correlates

There are several anatomical and neurophysiological correlates specific to Ia sensory fiber and alpha motor neuron connections occurring at the spinal cord level that would contribute to the predictions of sensorimotor integration as expressed by Marteniuk, MacKenzie, and Baba (1984) (Gordon & Ghez, 1991; Jankowska, 1992; Windhorst, Kokkoroyiannis, Laqu Coast, & Meyer-Lohmann, 1994). Figure 1 is a simplified illustration of the anatomical arrangement and connections between alpha motor neurons, interneurons, and Ia sensory fiber projections on a single and polysegmental level. As seen in Figure 1, motor output can influence the interneuronal pool, thus altering sensory input.

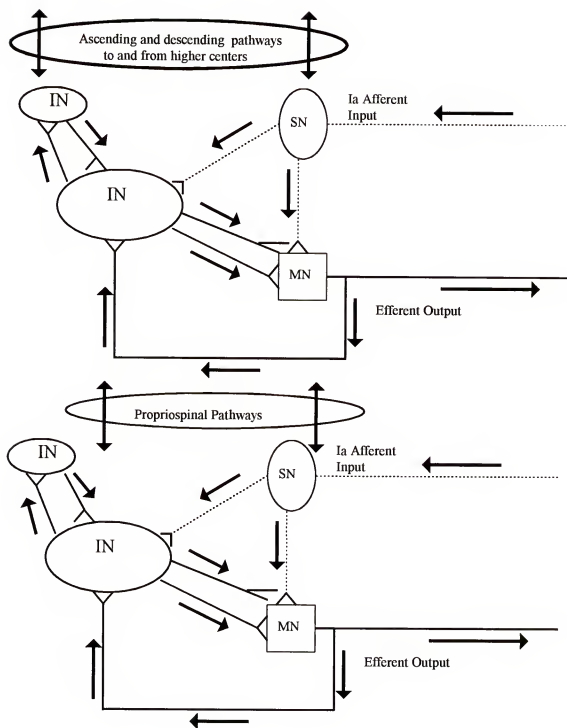


Figure 1. Simplified schematic of Ia sensory fiber connections with interneurons and motor neurons.

Conversely, sensory input can alter motor output, either directly through synaptic contacts with the alpha motor neuron or indirectly through the interneuronal pool.

In the event of polio pathology several possible scenarios exist that lead to predictions of altered sensory function, especially the sensory functions of Ia fibers. Based on the neuronal connections alone, a loss of motor neuron populations will alter the role of motor input to the interneuronal pool, which in turn modifies the input from the Ia sensory system. Another scenario involves the possible effect of transneuronal degeneration. Transneuronal degeneration refers to the process by which a neuron may undergo pathological changes and ultimately cellular degeneration when it loses its synaptic contacts (Duchen, 1992; Jessel, 1991; Willis & Grossman, 1973). Anterograde transneuronal degeneration refers to the loss of synaptic contacts which convey information to the cell whereas, retrograde transneuronal degeneration refers to the loss of synaptic contacts which convey information away from the cell (Jessel, 1991; Warwick & Williams, 1973).

In the case of polio, both anterograde and retrograde transneuronal degeneration may provide mechanisms that can effect sensory Ia fibers. Concerning anterograde degeneration, a loss of intrafusal muscle fibers within the muscle spindle would result in altered Ia stimulation, possibly resulting in cellular degeneration. Pathophysiologically, the muscle spindle neural connections can show degenerative changes in severe polio as evidenced by some spindle terminals becoming beaded and fragmented, particularly in severe disease (Carey, Massopust, Zeit, & Haushalter, 1944). The potential for retrograde degeneration is demonstrated by referring to Figure 1 and noticing the extensive synaptic contacts between the proximal projections of the Ia fiber to the motor neurons and

interneurons. Polio is recognized as primarily involving the neuronal cells of the anterior two-thirds of the spinal cord (Esiri & Kennedy, 1992). This distribution of involvement includes the cells to which the Ia sensory fibers have their primary projections (Guyton, 1991).

Sensorimotor integration provides a strong basis for predicting a deficit in Ia mediated sensory functions in individuals who have had polio. The reason for working within the theoretical perspective of sensorimotor integration stems from two distinct lines of reasoning. First, sensorimotor integration theory is well accepted in both the neurophysiological and psychophysiological literature (Brooks, 1986; Glencross, 1995; Marteniuk, 1992; Montgomery, 1991; Piek & Barrett, 1995). Secondly, sensorimotor integration provides a framework that is useful in identifying the importance of individual subsystem involvement while being able to account for the preservation of function. This is a particularly salient point for this investigation, as motor control is typically preserved in individuals who have had polio, which in all likelihood is the result of redundant systems and issues of motor equivalence (Marteniuk, 1992; Perry, Barnes, & Gronley, 1988; Requin, Semjen, & Bonnet, 1984; Turvey, 1990).

Sensorimotor integration was chosen as a theoretical backdrop after considering and rejecting several other accepted theoretical perspectives. The nature of the research question and the experimental methodologies used in attempting to answer this question were driving issues that contributed to the selection of a theoretical perspective. Theoretical perspectives that were contemplated ranged from reflex-based theories of motor control to more contemporary theories such as dynamical systems (Horak, 1991; Newton, 1995). Polio provides an interesting avenue of investigation into the

reorganization of the CNS following a primarily motor disease. This reorganization can be viewed from a cellular, spinal segmental level, or overt behavioral standpoint. Methodologies specific to describing changes at the cellular level and spinal segmental level are well identified in the basic biological sciences, and frequently use a reflex-based theoretical basis. However, approaching the global question of Ia mediated sensory functions required including an experimental approach which was more psychophysical in nature allowing the examination of joint position sense to perception. Approaching the question from a motor programming theoretical perspective was not considered appropriate because none of the experiments used in this investigation involved an examination of voluntary movement. Furthermore, the form of the question specified testing for dysfunction of a particular sensory system, without regard to learned motor behavior.

On the other hand dynamical systems theory presents an interesting perspective in that the system is viewed in total, and the adaptations of the system in response to the polio pathology are described in terms of self-organizing capabilities. However, the intent of this investigation was to determine the specific alterations of Ia mediated sensory functions. This in essence was a reductionist standpoint in which experimental methodologies were used to tease out the unique contributions of Ia system function without regard to the self-organizing capabilities of the entire system. From this standpoint, the question asked and the methodologies used were not consistent with the guiding philosophy of dynamic systems theory.

Adopting a sensorimotor integration theoretical approach to the question of sensory loss in individuals who have had polio provided a way to combine experimental

methodologies from both a psychophysical and neurophysiological perspective. Other theoretical tenants from a dynamical systems approach or a classical neurophysiological approach do not readily lend themselves to integrating psychophysical and neurophysiological experimental techniques. In the case of dynamical systems theory, the underlying supposition is that complex biological systems are interactive and self-organizing to maximize function. Thus, experimental paradigms which attempt to reduce complex behaviors into reduced main effects from specified subsystems provide little understanding of the complex observed behavior. On the other hand, neurophysiological findings of isolated subsystem failure, while descriptive in nature and certainly helpful from a clinical standpoint, often do not lead to an understanding of adaptations which allow function to be maintained. The use of sensorimotor integration as the theoretical basis for these experiments allowed the incorporation of differing experimental methodologies. This approach is supported by current trends recognizing the importance of a multidisciplinary approach in furthering the understanding of motor control (Glencross, 1995; Marteniuk, 1992; Piek & Barrett, 1995).

Polio

Pathophysiology of Acute Polio

Poliomyelitis (polio) is recognized as being an endemic disease within the human population present since early recorded medical history (Dalakas, 1995a; Laurie, 1990). Polio is the result of infection by one or more of three strains of polio virus, all classified as enteroviruses (Leestma, 1991). The virus enters the human host via ingestion, and initially infects the gastrointestinal tract. From the gastrointestinal tract the virus enters the blood stream, ultimately infecting the CNS. Evidence concerning the entry of the

polio virus into the CNS has implicated the myoneural junction of skeletal muscle as a portal along with the choroid plexus and capillary beds supplying spinal and autonomic ganglia (Carey, Massopust, Zeit, & Haushalter, 1944; Illa, Leon-Monzon, Agboatwalla, Dure-Samin, & Dalakas, 1995; Leon-Monzon, Illa, & Dalakas, 1995; Leestma, 1991). In approximately 90% of all polio infections the individuals will have only mild flu like symptoms without neurological signs, once referred to as abortive polio (Horstmann, 1949). Of the remaining 10%, a flu like non-paralytic disease often accompanied by signs of meningeal irritation occurs (Meiklejohn, 1952). The paralytic form of polio will occur in only 10-20% of those individuals who have the flu like symptoms, or only about 1-2% of all people infected with the polio virus (Fredericks, 1995; Leestma, 1991).

Once in the CNS the polio virus shows a predilection for attacking the anterior horn cells of the spinal cord, with an estimated 90-95% of the anterior horn cell population becoming infected (Dalakas, Bartfeld, & Kurland, 1995; Halstead, 1995). While no author refutes the severity of involvement of the anterior horn cell population, many investigators have shown that polio involves structures other than the anterior horn cell (Bodian, 1949; Bruno, Frick & Cohen, 1991; Bruno, Cohen, Galski, & Frick, 1994; Buchthal, 1949; Carey, Massopust, Zeit, & Haushalter, 1944). Bodian (1949) published a detailed account of the severity and distribution of polio induced lesions after studying 24 human autopsy cases. His findings showed that the polio virus can also infect cortical neurons primarily in the precentral gyrus, along with neurons in the hypothalamus, mid brain, reticular formation, vestibular nuclei, cerebellar nuclei, dorsal and intermediate horns of the spinal column, and spinal ganglia. More recently Bruno, Cohen, Galski, and Frick (1994) studied these central lesions in a group of long term polio survivors using

magnetic resonance imaging. Their findings confirm those of Bodian, as lesions were identified in the reticular formation, putamen, and medial lemniscus.

In acute paralytic polio, afflicted individuals will display fever and listlessness. These symptoms will progress to include stiffness of the neck, back and hamstrings along with muscle soreness and pain with attempted movement (Horstmann, 1949). Weakness is preceded by a change in deep tendon reflex responses to an initial hyperreflexia (increased reflexes) progressing to hyporeflexia (decreased reflexes) or areflexia (absent reflexes) (Mulder, 1995). Muscle weakness is typically asymmetrical, and is often more severe in the lower extremity muscles than in the upper extremity or trunk muscles (Nitshke, 1950; Fredericks, 1995). Additionally, the quadriceps followed by the tibialis anterior, and hip adductors are most frequently involved, with the quadriceps and hip adductors also being most severely involved (Nitshke, 1950). No definite sensory changes are noted with sensory examination (Forster, 1978). However, the report of pain with the occasional report of paresthesias (the sensation of numbness and tingling) may be indicative of sensory involvement (Bodian, 1949). The salient laboratory findings in acute polio include elevated cell and protein counts in the cerebral spinal fluid (Horstmann, 1949; Forster, 1978).

The pathophysiology of acute polio infection is rarely encountered in the medical practice of nations with vigorous immunization programs (Hodges & Kumar, 1986; Laurie, Maynard, Fischer, & Raymond, 1990; Streib, 1989). However, world wide, polio continues to be endemic with yearly epidemics, resulting in approximately 250,000 cases per year (Dalakas, Bartfeld, & Kurland, 1995). Treatment of acute polio focuses primarily on supportive and maintenance care, as no direct medical intervention will alter the

paralytic effects of acute polio (Forster, 1978; Horstmann, 1949). Medical management of the individual with acute polio focuses on maintaining respiratory, urinary and gastrointestinal system functions, protecting skin integrity, and relieving pain associated with muscle spasms and immobility (Forster, 1978; Fredericks, 1995; Horstmann, 1949). Once the acute aspects of the paralytic stage have subsided a detailed program of rehabilitation is imperative for maximizing functional return (Bennett, 1949). Though the widespread clinical understanding of polio was that it is an anterior horn cell disease, rehabilitation specialists recognized the potential for involvement of the sensory system, particularly those sensory mechanisms associated with proprioception and coordination (Bennett, 1949; Prior, 1960). Accordingly, rehabilitation efforts incorporated sensorimotor integration techniques into treatment plans (Bennett, 1949; Horstmann, 1949; Kabat, McLeod, & Holt, 1959; Prior, 1960). The ultimate outcome of acute paralytic polio reflects the severity of paralysis, the quality of acute and rehabilitative care, and the acceptance of patient limitations by themselves and their families (Bennett, 1949).

Pathophysiology and the Chronic Effects of Polio

The legacy of polio is far from a remote note of historical interest. Acute polio cases continue to be seen throughout the world, and the late effects of polio are becoming an increasingly recognized health problem (Halstead, 1995; Hodges & Kumar, 1986; Streib, 1989). The late effects of polio and their impact on health and function of polio survivors have been recognized in the medical literature since the late 1800's (Dalakas, 1995a). However, it has only been over the last decade that systematic and detailed studies of late polio pathophysiology have appeared in the literature (Agre, Rodriguez, & Sperling, 1989; Dalakas, 1988; Ravits, Hallett, Baker, Nilsson, & Dalakas, 1990;

Wiechers, 1988). In part, this increase in interest is driven by the increase in the number of polio survivors living to ages when the late effects of polio become pronounced, more than 30 years after the acute paralytic disease (Emeryk, Rowinska-Marcinska, Ryniewicz, & Hausmanowa-Petrusewicz, 1990; Frederick, 1995; Perry, 1988). With an estimated 1.63 million polio survivors living in the United States, and estimates ranging from 25-100% of all survivors developing late effects related to polio, post-polio sequela is second only to stroke as the leading cause of paralysis (Brummel-Smith, 1993; Bruno, Cohen, Galski, & Frick, 1994; Stone, 1994).

Agre, Rodriquez, and Tafel (1991) recommend using the term postpolio syndrome to describe the constellation of complaints and findings found in older polio survivors. These complaints include progressive weakness, fatigue, muscle pain, and joint pain are nonspecific in nature, and thus, postpolio syndrome is considered as a diagnosis of exclusion (Dalakas, 1995b). A subcategory of postpolio syndrome termed postpolio muscular atrophy is recommended by Dalakas (1988) when complaints of weakness are accompanied by findings of new muscular atrophy in muscles that were thought to be clinically stable or uninvolved. The pathophysiology of postpolio syndrome is thought to be multifactorial, encompassing both neuromuscular responses and functional etiologies (Agre, Rodriquez, & Tafel, 1991; Dalakas, 1995b).

The neuromuscular adaptations related to postpolio syndrome are being extensively studied using electrophysiological, histological, immunological, physiological, and magnetic resonance imaging techniques (Borg et al., 1989; Bruno, Frick, & Cohen, 1991; Lange, Smith, & Lovelace, 1989; Maselli, Cashman, Wollman, Salazar-Gruesso, & Roos, 1992; Rodriquez, & Agre, 1991). Electrophysiological studies cover a wide range

of applications including single motor unit analysis, macroelectromyography, power spectrum analysis, quantitative electromyography (EMG), and evoked potential testing (Grimby & Stalberg, 1995; Agre, 1995; Wiechers, 1995; Windebank, 1995). Histological studies are being used to study muscle fiber type changes, and the level of denervation and reinnervation occurring in stable and atrophied polio involved muscles (Borg & Edstrom, 1995; Dalakas, 1988; Drachman, Murphy, Nigam, & Hills, 1967). Immunological studies have shown that postpolio syndrome is not the result of new or latent virus activity, however, the possibility of an autoimmune response being in part responsible for postpolio syndrome continues to be investigated (Dinsmore & Dalakas, 1995; Melchers, et al., 1992). Physiological studies of persons with postpolio syndrome have shown decreased levels of growth hormone secretion, elevated levels of creatine kinase with exertion, and improved functional work capacity with modified training programs (Dean & Ross, 1988; Gupta et al., 1994; Warning & McLaurin, 1992). Magnetic resonance imaging studies have been used to evaluate the integrity of CNS structures and pathways as detailed by Bruno, Cohen, Galski, and Frick (1994), and in investigating peripheral muscular fatigue (Thompson, Barton, Marsh, & Cameron, 1991).

The picture of postpolio etiology is evolving by the integration of results from the studies listed in the above paragraph. A well-accepted finding is that the total number of available motor units is less in polio weakened muscle compared to non-involved muscle (Dalakas, 1995b; Daube, Windebank, & Lichy, 1995; Rodriguez, Agre, Black, & Franke, 1991). Additionally, these surviving motor units may be classified as normal (not affected), stable reinnervated (compensated but not normal), or unstable reinnervated (easily fatigued) (Wiechers, 1995). The stable reinnervated and unstable reinnervated motor units

both may show alterations in motor unit territory, motor unit amplitude, and altered onset and recruitment frequencies, along with abnormalities of the myoneural junction (Daube, Windebank, & Litchy, 1995; Maselli, Wollmann, & Roos, 1995). The surviving motor units in postpolio muscle take on the histochemistry of type I motor units as a result of adaptation to excessive use. However, the fiber typing based on immunohistochemical markers for fast or slow myosin isoforms show that this change is not the result of a selective loss of high threshold motor neurons (Borg et al., 1989; Borg & Edstrom, 1995). Morphologically, polio involved muscle shows a spectrum of changes, depending upon whether it was stable reinnervated or unstable reinnervated (Wiechers, 1995). Polio affected muscles which never returned to normal strength show both myopathic and neuropathic changes (dysfunction of both the muscle fibers and the nerve terminals), while those muscles which regained normal strength show a neuropathic pattern (dysfunction of the nerve terminals) of denervation/reinnervation changes (Dalakas, 1988; Drachman, Murphy, Nigam, & Hills, 1967).

Several studies have been conducted to determine differences between individuals who have had polio and those who have been diagnosed as having postpolio syndrome (Agre, Rodriquez, & Tafel, 1991; Dalakas, 1988; Ravits, Hallett, Baker, Nilsson, & Dalakas, 1990). These studies showed that those individuals with postpolio syndrome are weaker and require less time to fatigue (Agre, & Rodriquez, 1990; Rodriquez & Agre, 1991). However, neither chemical laboratory or quantitative and power spectral analysis of the EMG signal can differentiate between the two groups of polio afflicted individuals (Agre, Rodriquez, & Tafel, 1991; Dalakas, 1988; Maselli, Cashman, Wollman, Salazar-Gruoso, & Roos, 1992; Ravits, Hallett, Baker, Nilsson, & Dalakas, 1990; Rodriquez &

Agre, 1991). Bruno (1994) demonstrated differences in MRI imaging between groups of individuals with postpolio syndrome, such that those individuals having severe complaints of fatigue revealed an increase in the presence of lesions in the reticular activating system. Because of the lack of specific laboratory, electrophysiological, or imaging studies the diagnosis of postpolio syndrome remains a clinical decision of exclusion (Dalakas, 1995b; Windebank, 1995).

Currently, there is little research investigating the sensory consequences of postpolio. Most clinical standards specify that the clinical and electrophysiological sensory examinations are normal, and that abnormality would indicate an alternative pathology (Perry, Barnes, & Gronley, 1988; Windebank, 1995). However, the sensory consequences of postpolio would seem to be an area of fruitful research considering the well documented neuropathological evidence for lesions outside of the common final motor pathway in both acute and old polio, the presence of sensory complaints in acute and old polio, and the prediction of altered sensory function in response to motor dysfunction as derived from sensorimotor integration theory (Agre, Rodriquez, & Sperling, 1989; Bodian, 1949; Buchthal, 1949; Marteniuk, 1992; Prior, 1960). Perhaps most telling in this regard are the paraphrased observations made by Bodian (1949) concerning the relative absence of sensory findings “with severe cord involvement the intermediate and posterior horns may be damaged, in the presence of severe anterior horn damage flaccid paralysis is likely to be the only symptom. The possibility remains that internuncial neuron damage in the cord may contribute to the motor dysfunction of muscles not completely paralyzed” (p. 572).

Muscle Spindle

The muscle spindle is a motor and sensory organ that plays an important and multifaceted role in motor control (Brooks, 1986; Matthews, 1990; Schmidt, 1988). It functions as a sensory organ in that it provides direct sensory information related to muscle length and muscle length rate of change (Fredericks, 1995; Gordon & Ghez, 1991). This information is used in proprioception (Guyton, 1991; Martin & Jessel, 1991; Matthews, 1990; Winstein, & Schmidt, 1989). Additionally, the muscle spindle functions as a key component in maintaining and influencing spinal reflex drive (Hasan & Stuart, 1988; Matthews, 1990; Prochazka & Hulliger, 1983). The muscle spindle's role in proprioception and in reflex modulation is complex, and reflects the spindle's complicated neuromuscular anatomy which includes muscle fiber types with differing contractile properties and complex sensory and motor nerve innervation (Brooks, 1986; Warwick & Williams, 1973).

Anatomically the muscle spindle is situated in parallel with the extrafusal muscle fibers of its homonymous motor unit (Kimura, 1989). The muscle spindle is composed of 2 to 10 intrafusal muscle fibers surrounded by a connective tissue capsule. The connective tissue capsule of the muscle spindle connects to either the musculotendinous junctions or to the sides of an extrafusal muscle fiber (Fredericks, 1995). Furthermore, the intrafusal muscle fibers can be divided into either nuclear bag or nuclear chain fibers, with nuclear bag fibers having a tendency to have somewhat slower contractile properties (Gordon & Ghez, 1991).

The sensory innervation of the muscle spindle is rather complex containing two distinct types of sensory nerve fibers. The largest and fastest conducting sensory nerve

fibers, classified as group Ia fibers, connect to both the nuclear bag and nuclear chain intrafusal muscle fibers. A second type of sensory fiber, group II fibers, also connects to the muscle spindle, however, these are restricted to the nuclear chain intrafusal muscle fibers. While both the group Ia and group II sensory fibers are sensitive to dynamic and static stretch, the group Ia fibers fire more readily during dynamic stretch and the group II fibers are more sensitive to static stretch (Brooks, 1986; Gordon & Ghez, 1991).

The motor innervation of the muscle spindle is equally complex, as this entails two types of motor neurons, gamma and beta motor neurons, each subclassified into dynamic and static subdivisions. The dynamic gamma motor neuron fibers and static beta motor neuron fibers innervate nuclear bag intrafusal muscle fibers. The static gamma motor neuron fibers and dynamic beta motor neuron fibers innervate both the nuclear bag and nuclear chain intrafusal muscle fibers (Brooks, 1986; Guyton, 1991).

Physiologically the muscle spindle responds to the stimulus of stretch. This stimulus is then used by the central nervous system for proprioception and reflex modulation. The sensitivity of the muscle spindle to stretch is nonlinear, capable of responding to both dynamic and static components of stretch, and during both lengthening and shortening contractions (Brooks, 1986; Gordon & Ghez, 1991; Prochazka & Hulliger, 1983). These characteristics and their complex interrelationships have provided a fertile ground for research into how the muscle spindle functions as a proprioceptive organ and modulator of reflex activity (Boyd & Gladden, 1985; Matthews, 1990). Among the many tools used to study the muscle spindle and its physiological and behavioral output are proprioceptive positioning tests and tests of reflex excitability (Matthews, 1990; Schmidt, 1988).

Proprioception

Polymodal Aspects of Proprioception

Proprioception in its broadest sense is the awareness of the orientation of limbs to each other and the body, and the orientation of the body to the environment (Sherrington, 1906), and is a multimodal sensory event (Abbs & Winstein, 1990; Schmidt, 1988). Historically, the sensory modalities that mediate proprioception are be divided into two categories, the exteroceptors and the interoceptors. The category of exteroceptors includes vision, audition, and cutaneous receptors. These receptors take in information from the environment such as visual field changes, sound changes, and air temperature or air movement changes. This external environmentally generated information is then used to localize limb, body or limb-body positions. In comparison, interoceptors are stimulated by changes occurring inside of the body. Examples of interoceptors include the afferent sensory fibers of the muscle spindle (both type Ia and II), the Golgi tendon organ (type Ib), joint afferent fibers, and the vestibular apparatus. These fibers respond to either muscle length changes, muscle tension changes, joint position changes, and gravitational/acceleration changes (Guyton, 1991; Winstein & Schmidt, 1989). The evolution of such a complex and multimodal system directly supports the importance of sensory information, specifically proprioception, to motor control (Abbs & Winstein, 1990; Gordon, 1991).

The redundancy of subsystems within the sensory system that carry proprioceptive input explains the ability to maintain almost normal functions in the presence of disease or failure of one or more subsystems that modulate proprioception (Kelso, Holt, & Flatt, 1980; Sanes, 1990; Teasdale et. al., 1994). The very complex nature of proprioceptive influences makes it difficult to specifically measure the impact of any one subsystem on the

function of the system as a whole. This problem is readily demonstrated by the shifting attitudes concerning the muscle spindles' contribution to proprioception over the last decade.

Through the first-half of the century the muscle spindle was assumed to have great importance as a proprioceptor simply because of its anatomical arrangement, numerical prevalence, and the prevailing theory of reflexive control of movement. This notion was challenged by Lashley (1917) who provided evidence of intact active positioning capabilities in a person with a posterior column spinal cord injury. Additional challenges to the role of muscle spindle mediated proprioception occurred as a result of selective surgical procedures with removal of tendon attachments showing little impact on isolated joint positioning accuracy, and a lack of defined pathways to cortical centers (Smith, 1969; Warwick & Williams, 1973). However, technical advances in experimentation and developing neurophysiological techniques in the 1970s and 1980s have shown that the muscle spindle plays an important role in proprioception, influencing spinal functions, and contributing to the conscious perception of joint position awareness (Abbs & Winstein, 1990; Matthews, 1990; McCloskey, 1973).

1a Sensory Fiber Contributions to Joint Position Sense

While proprioception as a whole refers to the awareness of the limbs and body position both to themselves and the environment, the specific perception of joint movement is termed kinesthesia (Martin & Jessel, 1991). Additionally, the ability to accurately identify a specific stationary joint angle is referred to as joint position sense (McCloskey, 1973). In normal function, kinesthesia and joint position sense are mediated by both exteroceptors and interoceptors (Lackner & Taublieb, 1984; Paillard, &

Brouchon, 1974; Tardy-Gervet, Gilhodes, & Roll, 1986). However, in a normal sensory system, individuals retain a very high level of accuracy in the ability to judge joint position changes even when visual cues are eliminated (Berenberg, Shefner, & Sabol, 1987; Gandevia, McCloskey, & Burke, 1992).

The ability to further separate the respective roles of various interoceptors is difficult without surgical, medical or technological interventions. Surgically, the respective role of joint afferent and muscle spindle afferent function has been studied by pulling on exposed tendons, without actually moving the anesthetized joint (McCloskey, Cross, Honner, & Potter, 1983). In all participants, pulling the tendon resulted in the reporting of joint movement sensations in the direction of tendon stretch, for example pulling on a flexor tendon gave the sensation of joint extension. Further evidence for the role of muscle spindle mediated joint position sense is presented by studies in which the articular capsule is removed or anesthetized (Barrack, Skinner, Brunet, & Haddad, 1983; Kelso, Holt, & Flatt, 1980). Both interventions demonstrated remarkably preserved joint position sense function. While these findings by themselves are strong indications of the muscle spindles' role in joint position sense, the most definitive findings are derived from vibratory perturbation and microneurographic studies.

Vibratory perturbation. Vibratory perturbation as a method to study alterations in joint position sense, thought to be mediated via muscle spindle afferent fibers, has been extensively used over the last 25 years (Goodwin, McCloskey, & Matthews, 1972; Granit, 1972; McCloskey, 1973). Vibratory stimuli of amplitudes ranging from .5 mm - 2 mm, at frequencies of 80-120 Hz, induce altered sensory input such that illusionary movements occur and joint positioning accuracy is altered (Capaday & Cook, 1981; Inglis & Franks,

1990; McCloskey, 1973; Paillard & Brouchon, 1974). Specifically, vibration of a stationary muscle causes the illusion of movement in the direction that lengthens the tendon of the vibrated muscle, and this illusion is increased if the muscle isometrically contracts during the vibration (Craske, 1977; McCloskey, 1973; McCloskey, Cross, Honner, & Potter, 1983). Additional work has shown that the predominate error effect of vibration during active movement occurs when the antagonistic or lengthening tendon is vibrated versus the vibration of the agonistic or shortening tendon, and that this pattern of perturbation holds true for passive as well as active movements (Bullen, & Brunt, 1986; Capaday & Cooke, 1981; Inglis & Frank, 1990; Inglis, Frank, & Inglis, 1991).

Microneurography. The most direct form of measuring specific nerve fiber function in humans is microneurography. Microneurography requires that a very small electrode be inserted into a nerve fascicle, where the electrode then is able to record the individual action potentials generated along the nerve fiber (Prochazka & Hulliger, 1983). Microneurography by itself, and when coupled with vibratory perturbation has provided great insight into the firing patterns of muscle afferent fibers (Cordo, Gandevia, Hales, Burke, & Laird, 1993; Prochazka & Hulliger, 1983; Roll & Vedel, 1982). Using microneurographic techniques combined with vibration, Roll and Vedel (1982) revealed that illusionary movements increase with vibration frequencies up to 80 Hz. More detailed studies revealed that there is a great deal of selectivity for stimulation of Ia sensory afferents using low amplitude (.2-.5 mm) vibratory stimulation of frequencies up to 180 Hz (Cordo, Gandevia, Hales, Burke, & Laird, 1993). Furthermore, if done in a relaxed muscle this level of vibratory stimulation did not excite spindle type II afferents or Golgi tendon Ib afferent fibers to a significant degree (Roll, Vedel, & Ribot, 1989).

Vibratory perturbation and microneurography are teasing out the specific function of the Ia sensory fibers' role in joint position sense. However, this proprioceptive role is not the only function for Ia fibers, as the Ia fibers are a vital link in motor reflex arcs.

Muscle Reflex Modulation

Monosynaptic Reflex Arc

The monosynaptic reflex arc is an excellent model of sensorimotor integration (Gordon & Ghez, 1991). This two neuron system, a single motor neuron and a single sensory neuron, with only one connection or synapse between the neurons provides us with a remarkable opportunity for study. A summary of the reflex, shown in Figure 2, is that a quick stretch of the muscle spindle will stimulate the sensory receptors of the Ia nerve fibers. If stimulated to threshold, action potentials will travel along sensory fibers toward the spinal cord, there it will excite the homonymous motor neurons. The motor neurons will generate action potentials that travel to the muscle fibers making up its motor unit, and a contraction is observed (Gordon & Ghez, 1991; Guyton, 1991).

H-Reflex

The electrical analog to the muscle stretch reflex is the H-reflex (Fisher, 1992). This response is elicited by electrical stimulation of the Ia sensory fibers, as compared to the direct mechanical stretch of the muscle spindle (Fisher, 1992; Kimura, 1989). Though the muscle stretch reflex and the H-reflex share many similarities, they are not considered as equivalent responses as significant differences exist in the form of their stimulation and the extent of polysynaptic contributions to the evoked response (Burke, 1983; Burke, Gandevia, & McKeon, 1984). The H-reflex has been extensively used as an

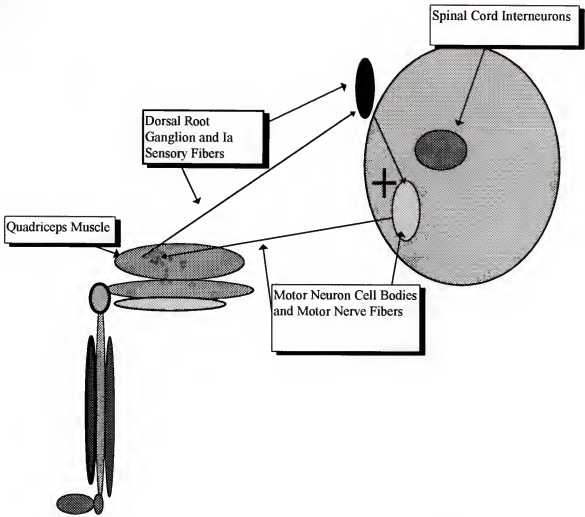


Figure 2. Neural pathways mediating the monosynaptic muscle stretch reflex from the quadriceps muscle.

electrophysiological tool in both clinical medicine and neurophysiological studies of motor control (Brooks, 1986; Goodgold & Eberstein, 1984; Kimura, 1989; Matthews, 1990).

From a neurophysiological standpoint the H-reflex is used in both animal and human studies as a method to assess motor neuron pool excitability, determine levels of presynaptic inhibition to Ia sensory fibers, and investigate spinal reflex pathways (Hultborn, Meunier, Morin, & Pierrot-Deseilligny, 1987; Schieppati, 1987; Taborikova & Sax, 1968).

A frequently used H-reflex measure is the Hmaximum/Mmaximum ratio. This represents the ratio between the maximum amplitude obtained during H-reflex testing and the maximum amplitude obtained during direct stimulation of the target muscle. This measure is most often used to infer motor pool excitability. However, multiple investigators question the assumption that H-reflex testing can be used to accurately measure entire motor pool excitability (Gerilovsky, Tsetinov, & Trenkova, 1985; Hultborn & Nielsen, 1995; Taborikova & Sax, 1968). The Hmaximum/Mmaximum ratio is a measure that has been used in comparing groups with and without pathology, and as a measure of excitability showing changes occurring with brain or spinal cord injury (Calancie et al., 1993; Faist, Mazevet, Dietz, & Pierrot-Deseilligny, 1994; Prakash, Sinha, Mukherjee, Katiyar, & Day, 1995).

Often the H-reflex is used as its own control value in experiments which attempt to show an alteration in motor pool excitability following some experimental manipulation. Typically the H-reflex will be expressed as a percent facilitation or inhibition of a control value H-reflex. This technique, while accepted in the literature, has generated

considerable controversy as experimental findings may be altered as the result of procedural or testing error, by confounding the experimental manipulation with size dependent changes of the test reflex (Crone et al., 1990; Hugon, 1973; Meinck, 1980; Kameyama, Hayes, & Wolfe, 1989)

Recent work has shown that the calculated result, inhibition or facilitation, as measured as a percentage of H-reflex control, depends on the amplitude of the control H-reflex (Crone et al., 1990; Meinck, 1980). That is, the magnitude of dependent variable changes are influenced by both the experimental manipulation and the level of the control H-reflex amplitude (Meinck, 1980). The logic of such findings can be explained by ceiling and floor effects similar to those noted in motor learning studies. In predicting a facilitation effect, the use of a high amplitude control H-reflex decreases the magnitude of the potential facilitation. Conversely, in studies predicting an inhibition effect, the use of a low amplitude control H-reflex decreases the magnitude of the potential inhibition. To minimize this problem, a control H-reflex value of 10-20% the M-wave maximum or a control H-reflex value of 40-50% the maximum M-wave is recommended for studies predicting a facilitation or inhibition effect respectively (Crone et al., 1990).

Closely aligned with the H-reflex control value is the issue of determining the proper calculation for the percent of facilitation or inhibition either after an experimental manipulation or between differing groups. Specifically, many studies have selected to use the control H-reflex value in the denominator when calculating the percent facilitation or inhibition, with the manipulated value of the H-reflex as the numerator. Because of the changing amplitude of the control H-reflex, this calculation procedure is questionable as it increases the variability of the measurement and the possibility of error. Additionally, if

the research question is to be related to the motor neuron pool activation, then the equation should reflect the motor neuron population and not a variable percent of that population as represented by the control H-reflex (Crone et al., 1990). More accurate calculations of percent facilitation or inhibition should reflect the M-wave maximum in the denominator of both measures. Thus, $H_{control}' = H_{control}/M_{maximum} \times 100$, and $H_{test}' = H_{test}/M_{maximum} \times 100$; so $H\Delta = H_{test}' - H_{control}'$. In this manner a negative number represents inhibition, a positive number represents facilitation, and both represent a percent referred to $M_{maximum}$.

H-Reflex Modulation

In spite of being sensitive to a variety of technique and methodological errors, the H-reflex is a powerful and reliable tool in studying the muscle spindle mediated reflex arc as it relates to clinical and theoretical concerns of human neurophysiology (Kimura, 1989; Matthews, 1990; McIlroy & Brooke, 1987; White, 1991; Williams, Sullivan, Seaborne, & Morelli, 1992). The H-reflex has become a corner stone in electrophysiologic measures designed to elucidate the spinal mechanism of sensorimotor control. Techniques such as homonymous voluntary facilitation, homonymous vibratory inhibition, and homonymous low frequency depression have been developed that enhance the scope of H-reflex studies.

Voluntary contraction facilitation. Voluntary facilitation is a term used to describe the facilitatory effect that voluntary contraction has on H-reflex amplitudes (Hultborn, Meunier, Pierrot-Deseilligny, & Shindo, 1987). The voluntary contraction is thought to increase the amplitude to the H-reflex response by (1) increasing voluntary drive, 2) decreasing presynaptic inhibition of the Ia fibers, and 3) increasing the gain of the muscle spindle making it more responsive to stretch (Bongiovanni, Hagbarth, & Stjernberg, 1990;

Hultborn, Meunier, Pierrot-Deseilligny, & Shindo, 1986; Nielsen, Sinkjaer, Toft, & Kagamihara, 1994; Schieppati, Nardone, & Musazzi, 1986). The utility of potentiating the H-reflex response by a volitional contraction is shown by this technique assisting in the diagnosis of neural pathology and by allowing recordings from muscles that typically will not generate H-reflex responses with traditional stimulating techniques (Burke, Adams, & Skuse, 1989; White, 1991).

Vibratory inhibition. Ia fibers are very sensitive to vibratory impulses. This sensitivity directly affects kinesthetic illusions, as well as producing a profound effect on the H-reflex. In the presence of vibration the H-reflex is markedly inhibited (Ashby, Stalberg, Winkler, & Hunter, 1987; De Gail, Lance, & Neilson, 1966; Desmedt & Godaux, 1978). This inhibitory effect occurs over both homonymous and heteronymous pathways, and is thought to be the result of presynaptic inhibition of the sensory Ia fibers (Ashby, Stalberg, Winkler, & Hunter, 1987; Hultborn, Meunier, Morin, & Pierrot-Deseilligny, 1987). Interestingly, vibration creates a paradoxical situation in that vibration, though inhibitory to the H-reflex, facilitates a physiological reflex muscle contraction known as the tonic vibration reflex (Desmedt & Godaux, 1978; Desmedt, 1983).

Low frequency inhibition. Low frequency depression or inhibition of the H-reflex refers to the reduction in amplitude of the H-reflex with stimulation frequencies between .1 and 10 Hz (Cook, 1968; Ishikawa, Ott, Porter, & Stuart, 1966; Van Boxtel, 1986). Low frequency depression of the H-reflex is generally attributed to presynaptic mechanisms, which may include a transient depletion of available neurotransmitter within the presynaptic terminal (Capek & Esplin, 1977; Cook, 1968; Van Boxtel, 1986). The

effects of low frequency depression can be modulated. Burke, Adams, and Skuse (1989) have shown that when coupled with voluntary contraction, the effects of low frequency depression are negated, even with stimulation frequencies up to 4 Hz.

TVR

The TVR is a low grade tonic involuntary muscle contraction that results from the application of a vibratory stimulus over the muscle belly or tendon (De Gail, Lance, & Neilson, 1966). This response is thought to be mediated over a polysynaptic pathway, requiring intact connections to higher centers (De Gail, Lance, & Neilson, 1966; Desmedt, 1983). As such the TVR is a reflex response that may be able to assist in differentiating between more selective involvement of Ia mediated responses over a monosynaptic or polysynaptic pathway.

Summary

A theory of sensorimotor integration provides a broad based framework in which to study normal and abnormal movement control (Burtner, & Woollacott, 1995; Grime, 1983; Pickenhain, 1984; Phillips, Muller, & Stelmach, 1989; Phillips & Stelmach, 1992). The theoretical framework allows for the development of predictions concerning altered movement system functions in response to different models of pathology. In particular, sensorimotor integration specifies that deficits in sensory systems may in altered function of the motor system, and conversely deficits in motor systems may result in altered function of the sensory system. Polio, a primary motor disease, is the avenue for investigating a specific motor disease that potentially alters sensory function. The muscle spindle is identified as a dynamic sensorimotor structure, having both afferent and efferent neural connections. Based on these connections the muscle spindle, which mediates

functions related to joint position sense and reflex modulation, is considered a physiological testing ground for sensorimotor integration (Gordon & Ghez, 1991). Both joint position sense and reflex modulation rely on appropriate functioning of the Ia sensory fiber. Selective experimental manipulations designed to isolate Ia function in joint position sense and reflex modulation using vibratory perturbation and electrical stimulation have the capability to delineate Ia sensory fiber mediated functions. Combining these experimental manipulations and testing polio as a model of primary motor disease will provide evidence for Ia mediated sensory changes as a result of motor disease as predicted sensorimotor integration.

CHAPTER 3 METHODS

Participants

Participants ($N = 30$) were adult males and females, with 15 of them having had polio while the remaining 15 served as controls having no history of prior poliomyelitis (see Appendix A for sample size calculations). Inclusion criteria for those who have had polio was that the tested quadriceps muscle strength be rated no greater than a grade of 'good' and no less than a grade of 'poor' by manual muscle testing.

Participants were matched by age to within ± 5 years. All were free from other neurological, orthopedic, and medical disorders which may have interfered with their ability to participate in this study. A brief medical history and neuromuscular screen was conducted to insure that participants met inclusion criteria and had no cutaneous sensory deficits indicative of other possible confounding pathologies (see Appendix B). Additionally, they completed the digit symbol substitution test (Form 1) as a screening effort for memory deficits (Gilmore, Royer, & Gruhn, 1983; Salthouse, 1992). Participants were recruited through local polio support groups, social organizations, and the Center for Exercise Science, University of Florida. They all completed the approved human subject consent form prior to their participation (see Appendix C).

Apparatus

Joint position sense testing was accomplished using an electrogoniometer (Therapeutics Unlimited) and an amplifier module (Therapeutics Unlimited). Knee motion

speed was controlled by an electrical dynamometer (Cybex II). Vibratory perturbation was provided by a vibrator/exciter (Bruel & Kjaer, model #4809) amplified by a 100 watt power amplifier (Radio Shack) and frequency controlled by a function generator (Wavetek). Linear excursion of the vibration was measured on line by a linear velocity detector (Macro Sensors), powered by a 15 V transformer (Sola). All data acquisition and intermediate analyses were conducted on Data Pac software (Run Technologies), with a sampling rate of 200 Hz.

The EMG instrumentation used in the stimulation and collection of H-reflex responses was composed of multiple subunits. The surface recording electrodes (Therapeutics Unlimited) were Ag-Ag/Cl housed in a plastic case which also contains circuitry for the preamplifier, which amplifies the EMG signal on site by 35x. The EMG recording electrodes were connected to high impedance (15m Ω at 100 Hz) amplifier units (Therapeutics Unlimited), having a bandpass width of 20-4000 Hz, an adjustable gain setting set to 1000x, and a common mode rejection ratio of 87 dB at 60 Hz.. The analog EMG signal was converted into a digitized format and stored to the hard drive of a Zeos microcomputer for later analysis. Electrical stimulation was provided by a dual channel (Grass 88) electrical stimulator along with stimulus isolation (Grass SIU 5) and constant current control sources (Grass CCU 1). Real time EMG and evoked potential waveforms were displayed on an oscilloscope (Tektronix TDS 310). All data acquisition and analyses was conducted on Data Pac software (Run Technologies), with a sampling rate of 20 kHz.

Procedures

Prior to testing, all participants read and signed the approved human subject consent form, and were given ample opportunity to ask questions concerning the purpose,

procedures, and risks associated with this study. They were given a neuromuscular screening exam of the lower extremities including manual muscle testing, cutaneous sensory testing, tendon reflex testing, and range-of-motion testing.

Joint Position Sense

Participants were seated on a padded chair with their hips in 90° of flexion and their knees in 80° of flexion. An electrogoniometer was firmly taped and elastic wrapped to the medial aspect of the involved leg. Additionally, EMG electrodes were firmly taped to the vastus medialis muscle with double sided adhesive tape and elastic wrap. The involved lower extremity was loosely secured across the thigh to the padded chair, and loosely secured by the lower leg to the moment arm of the dynamometer. The dynamometer was adjusted such that the speed of rotation is 3° per s. Participants were given a signal buzzer to hold in the hand of their choice. The vibrator was positioned over the patellar tendon, just proximal to the patella (see Figure 3).

Each participant received five practice trials in the following manner. With the eyes shut, the involved limb's knee was passively moved to a position somewhere between 30° and 60° of knee flexion. Once this position was attained, the knee was held stable for 3 s by the experimenter. The participant was verbally prompted to remember this position, and they acknowledged their understanding by pressing a hand-held buzzer. Once the buzzer was pressed the knee was passively moved into extension for a distance between 10° and 30° from the target position. After a time delay, between 5 and 10 s, the participant's knee was allowed to passively move into flexion and when the knee returned to the criterion position, participants pressed the buzzer switch. After five practice trials at different testing angles, formal testing began.

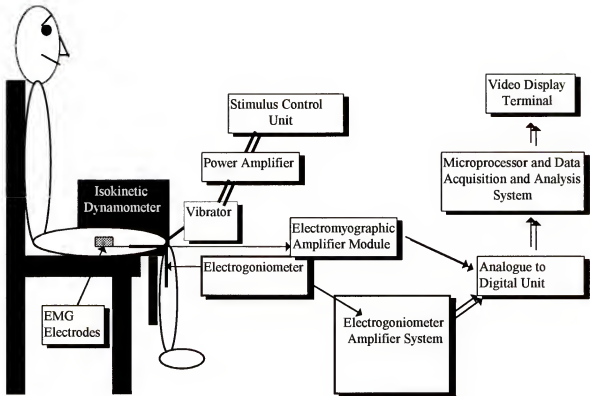


Figure 3. Knee joint position matching task instrumentation showing vibrator, EMG, electrogoniometry, and dynamometry control units.

During formal testing, each participant was given two blocks of 10 trials of the knee joint positioning task, one with and one without vibratory perturbation. Testing mimicked the practice trials with the only difference being that on one block of 10 trials the quadriceps tendon will be vibrated during the repositioning task. The vibration frequency was 100 Hz, with an amplitude excursion of .75 - 1 mm. Vibratory perturbation started right before participants began a repeat positioning task trial, and lasted throughout the trial. The knee position target angles, distance moved to targets, and time delay between repositioning tests was similar between participants with the order randomly presented (see Appendix D). A 30 s rest was given between trials. The order of testing with or without vibration was counterbalanced between participants (see Appendix D). In addition, the EMG activity of the vastus medialis muscle was monitored to ensure that all testing was administered in a passive manner without active contraction of the quadriceps muscle, and that the vibration did not induce a tonic vibratory reflex.

H-Reflex

The four H-reflex experiments were conducted with participants resting in a supine position on a padded table with a pillow beneath their head. Care was taken to ensure that the participant's head was in neutral rotation, arms relaxed, and legs extended in a comfortable position. Prior to testing, the skin over vastus medialis muscle, femoral triangle, and proximal posterior thigh was prepared and cleaned with isopropyl alcohol. An EMG active electrode coated with conductive paste was fixed over the vastus medialis muscle with adhesive fitted pads and reinforced with elastic wrap and tape.

The stimulating cathode for the femoral nerve was positioned slightly lateral to the femoral artery and just distal to the inguinal ligament over the proximal anterior thigh.

The stimulating anode was positioned in the midline of the proximal posterior thigh just distal to the gluteal fold (see Figure 4). Both electrodes were coated with conductive paste and taped in place, with increased pressure over the cathode provided by a moldable pad secured in place by a pelvic belt. A ground electrode coated with conductive paste was taped and wrapped in place over the medial aspect of the proximal tibia.

The four different experiments were conducted using different forms of H-reflex manipulation. For all participants, the first test was to determine the maximum values for the direct evoked compound muscle (M-wave) action potential and for the reflex evoked H-reflex. The remaining three experiments are named: (a) voluntary facilitation, (b) vibratory inhibition, and (c) low frequency inhibition. To control for potential test order effects, the presentation sequence for these last three experiments was randomly presented across participants (see Appendix D).

M-wave and H-reflex maximum comparisons. The procedure for determining maximum amplitude values for the M-wave and H-reflex involved a stepwise progression of increasing voltage until no further gain in amplitude was observed. Stimulation rate will be set at 1 every 10 s. Pulse duration will be set at 1 ms. The voltage was gradually increased, approximately in 10 volt steps, throughout the range of initial evoked response to maximum M-wave value. Fine tuning of the electrical stimulation was conducted at the maximum H-reflex and M-wave response values to insure that the response was of maximum value. Approximately 20 stimulations were required for this testing procedure.

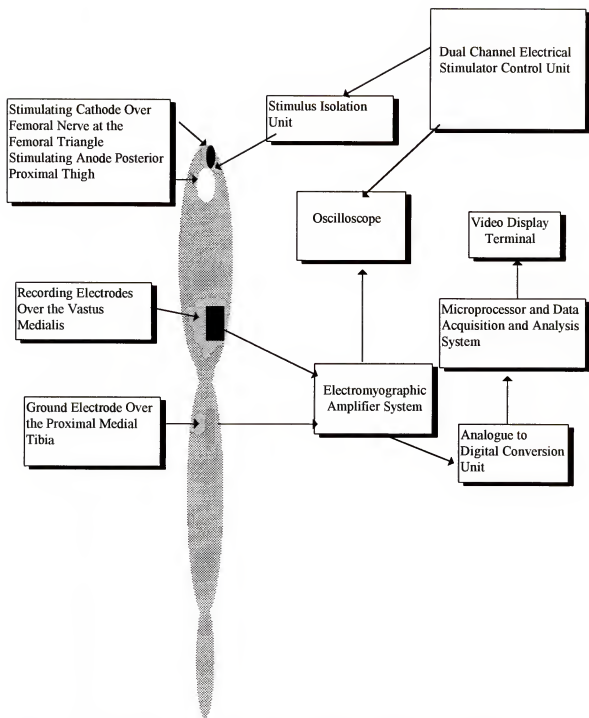


Figure 4. Equipment set-up and participant circuit for H-reflex recordings from the quadriceps.

Voluntary facilitation. Homonymous voluntary facilitation involved an adaptation of the experimental set-up such that the participant triggered the electrical stimulator by generating a quick, low intensity, voluntary contraction of the target muscle (vastus medialis). For establishing control values, the femoral nerve was stimulated by an electrical impulse of 1ms duration, once every 10 s for a total of 10 trials. Control H-reflex values were collected with stimulation intensities such that the H-reflex amplitude was approximately 10-20% of the maximum M-wave amplitude. Ten control value H-reflexes were recorded.

Voluntarily facilitated responses were generated by the participant generating a quick low intensity voluntary contraction of the vastus medialis. This contraction was just palpable to the touch, and was recorded by the EMG electrodes over the vastus medialis. As shown in Figure 5 (shaded areas represent equipment changes) the EMG signal was amplified and transmitted to a marker pulse generating unit. If the impulse was of threshold value, the marker pulse generating unit triggered the electrical stimulator, and an H-reflex was induced and recorded. Stimulation parameters were constant as during the control testing period. The participant generated a total of 10 voluntary facilitated H-reflex recordings, at a rate of 1 every 10 s.

Vibratory inhibition. For this experiment, inhibition of the H-reflex was studied by using a vibratory stimulation. Ten control value vastus medialis H-reflexes were recorded with the electrical stimulator set at a pulse rate of 1 every 10 s., with a pulse duration of 1 ms, and the stimulus intensity adjusted such that the H-reflex amplitude was approximately 10-20% of the M-wave maximum value. Ten vibration perturbed H-reflexes were recorded using the same electrical stimulator settings. The vibratory perturbation was

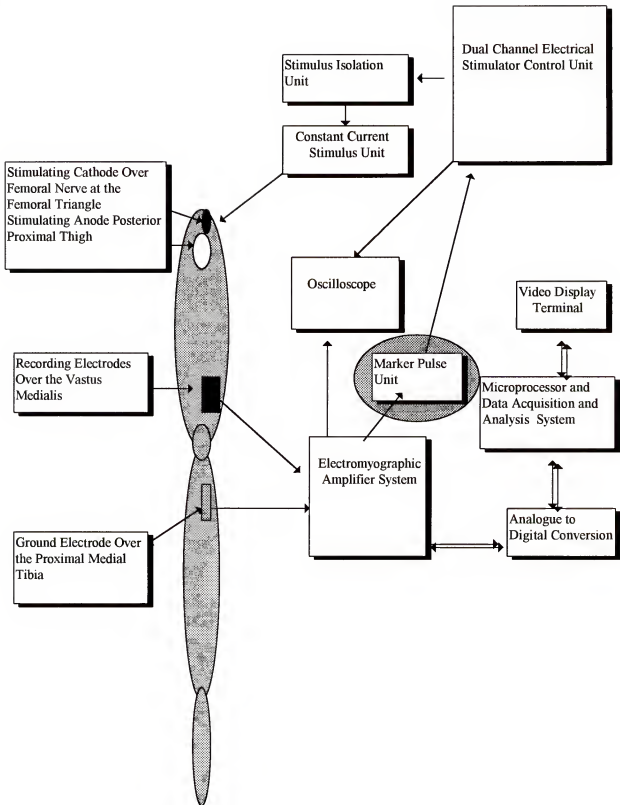


Figure 5. Equipment set-up and participant circuit for H-reflex recordings from the quadriceps during homonymous voluntary facilitation.

delivered at a rate of 100 Hz for a sequence of 10 cycles. On the 7th vibration cycle the electrical stimulator was triggered. Vibration was applied to the patellar tendon, slightly proximal to the patella (see Figure 6).

Low frequency inhibition. The equipment setup for this experiment was identical to that illustrated in Figure 4 with the addition of the constant current control unit. The manipulated variable was frequency of electrical stimulation. Ten control value vastus medialis H-reflexes were be recorded with the electrical stimulator set at a stimulation frequency of .1Hz, with a pulse duration of 1 ms, and the stimulus intensity adjusted such that the H-reflex amplitude was approximately 10-20% of the M-wave maximum value. These were followed by recording 10 test reflexes, recorded with the electrical stimulator set at a frequency of 1Hz and 5Hz.

TVR

The tonic vibration reflex experiment was conducted with the participant in the seated position with the involved knee in 70°-80° of flexion. The vibrator was positioned over the quadriceps tendon proximal to the patella. Pressure of the vibrator over the tendon was adjusted to approximately 22N. The vibrator frequency was set at 100Hz, with an amplitude excursion of 2mm. Prior to vibration the resting level EMG response was recorded from the vastus medialis for 10s. The vibrator was then turned on, and the EMG activity from the vastus medialis was recorded for 60s.

Experimental Design and Analyses

Descriptive data for the experimental and control groups included age, gender, and scores from the digit symbol substitution test. Additionally, quadriceps muscle strength and deep tendon response grades were recorded.

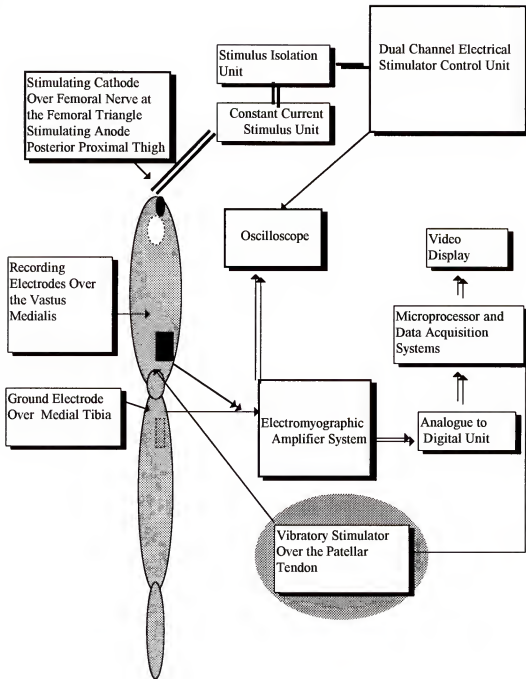


Figure 6. Circuit for H-reflex recordings from the quadriceps during homonymous vibratory inhibition.

The dependent variables for the joint-position matching task included mean CE (CE represents a measure of average error), mean ACE (ACE represents the absolute value of CE), and AE (AE measures the overall accuracy of performance) in degrees (Schmidt, 1988). Measurement error scores such as CE, ACE, and AE have been used extensively in motor control testing of individuals with and without pathology (Haaland & Harrington, 1989; Schmidt, 1988). Separate analyses were conducted on each dependent measure using a mixed design 2 x 2 (Group x Vibratory Condition) ANOVA with repeated measures on the vibratory condition factor.

Dependent variables for the H-reflex experiments included M-wave maximum amplitude (mV), H-reflex maximum amplitude (mV), Hmaximum/Mmaximum ratios, M-wave latencies (ms), H-reflex latencies (ms), level of facilitation, and level of inhibition calculations. Descriptive statistics for the dependent measures from the H-reflex experiments included Hmaximum/Mmaximum means, standard error of the mean, and ranges. Data for the Hmaximum/Mmaximum ratio experiments were analyzed with one-tailed paired *t*-tests. The data from the low frequency inhibition, vibratory inhibition, and voluntary contraction facilitation were analyzed in mixed design ANOVAs.

Data from the tonic vibration reflex were descriptive in nature. Resting level EMG was recorded for 10s. It was intentioned that the EMG activity associated with the tonic vibration reflex was to be described during the last 10s of the vibration, and expressed as a percentage increase above the resting level EMG.

CHAPTER 4 RESULTS

Descriptive Data Analyses

Thirty-nine individuals were recruited to participate in this study. Only the data from 30 individuals were used for data analyses as nine individuals were excluded; four because they did not meet experimental inclusion criteria, three were unable to tolerate experimental procedures, and two because of technical difficulties. Fifteen participants had a history of polio while the remaining 15 served as controls, age matched to within 5 years. Descriptive data for the groups are displayed in Table 1. Graded quadriceps muscle strength and quadriceps deep tendon responses for each group are shown in Table 2. Additionally, all participants completed the digit symbol substitution test as a screening tool for memory abilities. The mean score for the polio group was 48.9 ($SD = 10.11$) and for the control group 56.3($SD = 8.22$).

Table 1

Age and Gender Characteristics for Polio and Non-polio Participants

<u>Group</u>	<u>Age</u>		<u>Gender</u>	
	Mean (Years)	Range(Years)	Female(N)	Male(N)
Polio	55.2	40 - 75	5	10
Non-polio	55	36 - 74	8	7

Table 2

Ranked Scores for Quadriceps Muscle Strength and Deep Tendon Response

<u>Group</u>	<u>Muscle Strength</u>	<u>Tendon Response</u>
	Grade 2 3 4 5	0 1 2 3
Polio	1 6 8 0	10 5 0 0
Non-polio	0 0 0 15	0 3 9 3

Preliminary AnalysesJoint Position Sense

All data were stored directly to the microcomputer's hard drive, and subsequently backed-up to tape cassettes. All trials for the joint positioning sense and H-reflex experiments were marked manually for measurement. For the joint positioning sense experiments, voltage measures were taken for the criteria and test trials in both the no vibration and vibration conditions. Differences were calculated by subtraction of the criterion trial from the test trial. All voltage differences were converted to degrees using the formula: Degrees = Volts $\times$ 1°/.03V. Further calculations were carried out by converting the raw scores into error scores of CE, ACE, and AE. For each condition a total of 10 raw scores were used for each participant. Table 3 provides the calculations for determining the three error scores for the joint positioning experiment.

H-Reflex Experiments

The M-wave and H-reflex data were collected by visual inspection and manual marking of the evoked response traces. Takeoff latencies (ms) were identified as the first

Table 3

Formulas for CE, ACE, and AE

Formula for CE	$\frac{\sum_{i=1}^n (x_i - T)}{n}$
Formula for ACE	$ CE $
Formula for AE	$\frac{\sum_{i=1}^n x_i - T }{n}$

visual deflection from the baseline for both the M-wave and H-reflex. Amplitudes were recorded in volts (V) and defined as the difference between the greatest positive peak and the greatest negative peak for both the M-wave and H-reflex. Table 4 lists the formulas for calculating Hmaximum/Mmaximum ratios, level of inhibition, and level of facilitation. In the experiments studying facilitation or inhibition, data analyses were conducted using the mean H-reflex value of 10 trials expressed as a percentage of the M-wave maximum for the control and test conditions.

Primary AnalysesJoint Position Sense

Descriptive and inferential statistics for the joint positioning sense experiments are presented for the joint position sense experiments. The three dependent variables (CE,

Table 4

Formulas for H-reflex Calculations

H/M maximum	$\frac{H_{\text{maximum}}}{M_{\text{maximum}}}$
Inhibition	$\frac{H_{\text{test}}}{M_{\text{maximum}}} - \frac{H_{\text{control}}}{M_{\text{maximum}}}$
Facilitation	$\frac{H_{\text{test}}}{M_{\text{maximum}}} - \frac{H_{\text{control}}}{M_{\text{maximum}}}$

ACE, and AE) were analyzed with separate 2 x 2 (Group; polio and non-polio x Condition; vibration and no vibration) mixed design ANOVAs. For all analyses the level of significance was set at .05

CE. Descriptive statistics for the mean CE data are presented in Table 5 (all tables with descriptive statistics are located at the end of this chapter). Analysis of the CE data revealed a significant Group x Vibration Condition interaction, $F(1, 28) = 17.92, p < .01$. Figure 7 is a graphic illustration of the interaction effect, showing the non-polio group having a strong response to vibration by undershooting the target while the polio group actually tended to overshoot the target during the vibration condition. Additionally, a significant main effect was found for the vibration condition, $F(1, 28) = 15.87, p < .01$. This effect is shown in Figure 8.

ACE. Descriptive statistics for the ACE data are presented in Table 6. The only significant ACE finding identified was a main effect for the vibration condition, $F(1, 28) = 7.07, p < .02$. Figure 9 graphically displays this main effect; during the vibration condition both groups increased their ACE compared to the no vibration condition

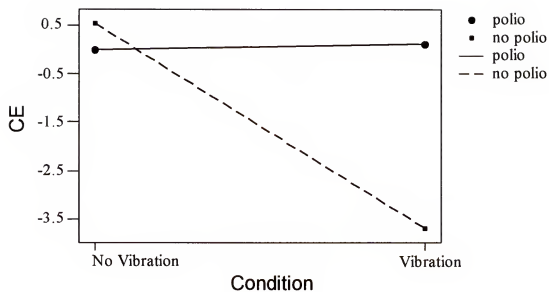


Figure 7. Group x Vibratory Condition for CE.

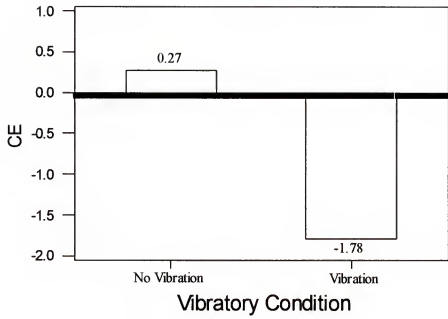


Figure 8. Vibratory main effect for CE.

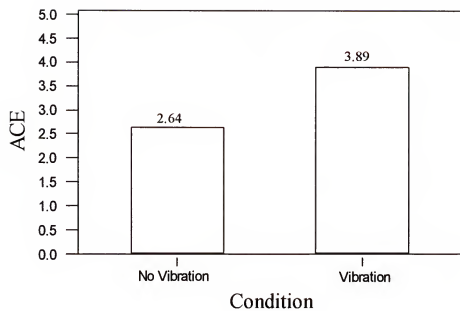


Figure 9. ACE vibratory main effect.

AE. Descriptive statistics for the AE data are presented in Table 7. The AE data closely parallels the CE scores, revealing a significant Group x Vibration Condition interaction, $F(1, 28) = 4.38, p < .05$. The interaction effect indicated a strong response to vibration in the non-polio group (see Figure 10) resulting in an increase of the AE score in comparison to the polio group. As with the CE data, the AE data also showed a main effect for the vibration condition, $F(1, 28) = 12.1, p < .01$. As illustrated in Figure 11 the vibration condition shows a greater value for AE than the no vibration condition.

H-Reflex Experiments

Only data from those participants who could tolerate the electrical stimulation, and whose evoked responses maintained a stable baseline, was used in the H-reflex analyses. Subsequently, each of the four H-reflex data bases are made up of different pairs of participants. Mean data for the Hmaximum/Mmaximum ratio experiment was analyzed with a one-tailed paired *t*-test. However, separate mixed design ANOVAs were used to evaluate the low frequency inhibition, vibratory inhibition, and voluntary contraction data.

Hmaximum/Mmaximum ratio. The data for the Hmaximum/Mmaximum ratio experiments were from 28 participants. Two participants were excluded for not being able to tolerate maximal levels of electrical stimulation. The descriptive statistics for mean, range, and SEM are presented in Table 8 for both the polio and non-polio groups. A one-tailed paired *t*-test demonstrated the significant difference between the two groups, $t(13) = 2.55, p < .02$. The bar graph in Figure 12 clearly shows the non-polio group having a larger Hmaximum/Mmaximum ratio as compared to the polio group.

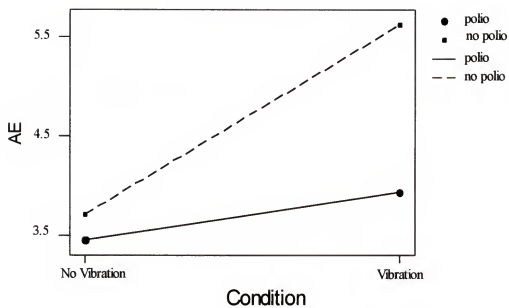


Figure 10. Group x Vibration Condition for AE.

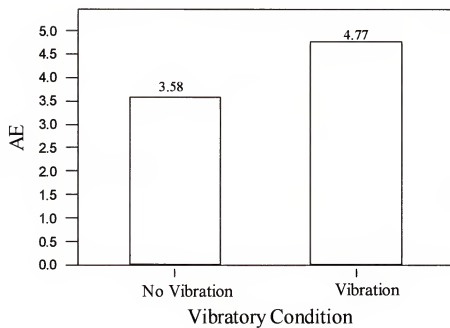


Figure 11. AE for vibratory main effect.

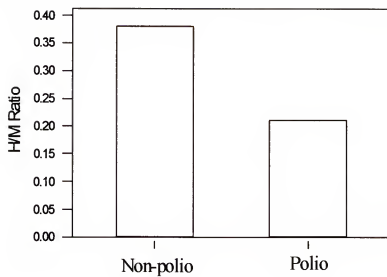


Figure 12. Hmaximum/Mmaximum ratios for the non-polio and polio groups.

Low Frequency Inhibition. The low frequency inhibition data set is for 10 participants. This number is the result of one member of six different participant pairs from 14 participant pairs having no identifiable H-reflex. Further attrition of three pairs occurred because of unstable responses, and technical difficulties. Table 9 displays the descriptive statistics for this experiment. The 2×3 (Group; polio and non-polio x Stimulation Frequencies; .1Hz, 1Hz, and 5Hz) mixed design ANOVA did not reveal any significant findings. Figure 13 is presented to illustrate the data trend for the group responses to the three stimulation frequencies.

Vibratory Inhibition. The vibratory inhibition scores were derived from the responses of six pairs of participants. Other data pairs from the 14 original pairs were excluded as the result of having no identifiable H-reflex (6 pairs), and because of technical difficulties with data recording (2 pairs). The descriptive statistics for the vibratory inhibition experiment are given in Table 10. A 2×2 (Group; polio and non-polio x Vibratory Condition; vibration and no vibration) mixed design ANOVA did not identify a significant interaction or main effect as illustrated in Figure 14.

Voluntary Contraction Facilitation. Data for 10 pairs of participants qualified for inclusion in the voluntary contraction facilitation analysis. Table 11 lists the descriptive statistics by group for the voluntary contraction facilitation experiment. A 2×2 (Group; polio and non-polio x Voluntary Contraction Condition; no contraction and contraction) mixed design ANOVA failed to yield a significant interaction effect. However the analysis did yield a significant main effect for contraction condition, $F(1, 18) = 30.38, p < .01$. Figure 15 is presented to graphically display the trend in the voluntary contraction facilitation data.

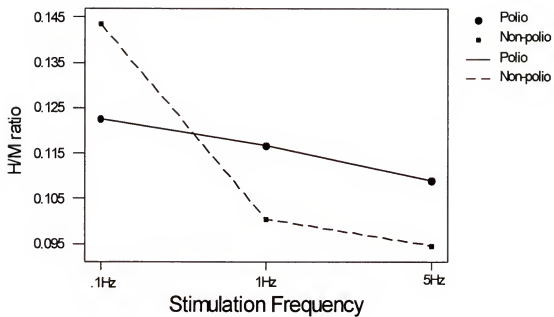


Figure 13. Percent H/Mmaximum ratios with low frequency stimulations of .1Hz, 1Hz and 5Hz for the polio and non-polio groups.

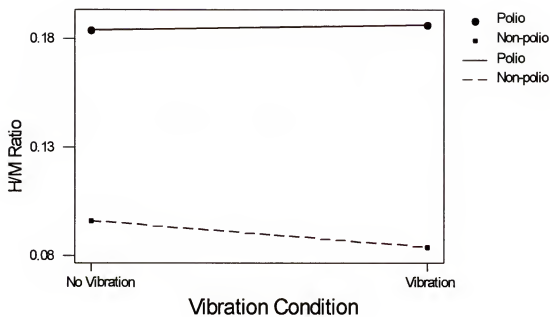


Figure 14. Vibratory inhibition for the polio and non-polio groups in response to 100Hz vibration.

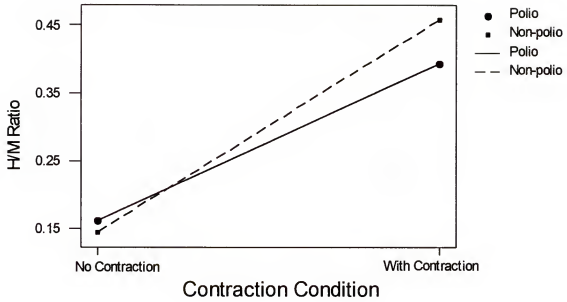


Figure 15. Voluntary contraction facilitation of the H-reflex for the polio and non-polio groups.

TVR

The tonic vibration reflex experiment did not yield useful information, and it was discontinued after being attempted on 17 participants. Of them only two (one from each group) demonstrated a tonic vibration reflex using the stimulation parameters of this experiment. This lack of effect, particularly in the non-polio group, indicated that the stimulation characteristics used for this experiment were not effective in eliciting the normal physiologic response.

Summary

In summary, the joint position sense analyses revealed a significant Group x Vibratory Condition interaction for both CE and AE. This interaction indicated that individuals who had polio showed no or a markedly less degree of undershooting in their response accuracy during vibratory perturbation than did individuals who had no history of polio. The ACE analyses revealed a significant main effect for condition, indicating that with vibration there was greater ACE during vibration for both groups.

Analyses of the Hmaximum/Mmaximum ratios indicated that the non-polio group had a significantly higher ratio than the polio group. However, separate analyses of the low frequency inhibition, vibratory inhibition, and voluntary contraction facilitation experiments did not identify any significant interaction effects between groups and conditions. The voluntary contraction facilitation experiment did reveal a significant main effect for condition, with both groups demonstrating an increase in Hmaximum-/Mmaximum ratios with voluntary contraction.

Table 5

Descriptive Statistics for CE

<u>Group</u>	<u>Condition</u>	<u>Mean</u>	<u>Range</u>	<u>SEM*</u>
Polio	No Vibration	-.002	-3.83 - 8.16	.815
	Vibration	.127	-6.36 - 7.2	.915
No Polio	No Vibration	.544	-9.06 - 7.73	.998
	Vibration	-3.70	-9.50 - 4.47	1.07

*SEM = Standard error of the mean

Table 6.

Descriptive Statistics for ACE

<u>Group</u>	<u>Condition</u>	<u>Mean</u>	<u>Range</u>	<u>SEM*</u>
Polio	No Vibration	2.35	.133 - 8.16	.518
	Vibration	2.82	.400 - 7.20	.518
No Polio	No Vibration	2.94	.400 - 9.06	.632
	Vibration	4.92	.867 - 9.50	.608

*SEM = Standard error of the mean

Table 7.

Descriptive Statistics for AE

<u>Group</u>	<u>Condition</u>	<u>Mean</u>	<u>Range</u>	<u>SEM*</u>
Polio	No Vibration	3.45	2.16 - 8.16	.394
	Vibration	3.92	2.10 - 7.20	.406
No Polio	No Vibration	3.70	1.73 - 9.06	.537
	Vibration	5.62	2.36 - 9.50	.547

*SEM = Standard error of the mean

Table 8.

Descriptive Statistics for Hmaximum/Mmaximum Ratios

<u>Group</u>	<u>Mean</u>	<u>Range</u>	<u>SEM*</u>
Polio	.21	0 - .69	.05
No Polio	.38	0 - .84	.05

*SEM = Standard error of the mean

Table 9.

Descriptive Statistics for Low Frequency Inhibition

<u>Group</u>	<u>Condition</u> <u>Stimulation Frequency</u>	<u>Mean</u>	<u>Range</u>	<u>SEM*</u>
Polio	1/10s	.12	.03-.24	.03
	1/s	.11	.03-.22	.03
	5/s	.10	.003-.19	.03
No Polio	1/10s	.14	.01-.23	.04
	1/s	.10	.02-.18	.03
	5/s	.09	.001-.17	.03

*SEM = Standard error of the mean

Table 10.

Descriptive Statistics for Vibratory Inhibition

<u>Group</u>	<u>Condition</u>	<u>Mean</u>	<u>Range</u>	<u>SEM*</u>
Polio	No Vibration	.18	.02-.47	.07
	Vibration	.18	.02-.46	.08
No Polio	No Vibration	.09	.02-.19	.02
	Vibration	.08	.01-.18	.03

*SEM = Standard error of the mean

Table 11.

Descriptive Statistics for Voluntary Contraction Facilitation

<u>Group</u>	<u>Condition</u>	<u>Mean</u>	<u>Range</u>	<u>SEM*</u>
Polio	No Contraction	.16	.00-.32	.03
	Contraction	.39	.00-.85	.09
No Polio	No Contraction	.14	.02-.32	.02
	Contraction	.45	.09-.77	.06

*SEM = Standard error of the mean

CHAPTER 5

DISCUSSION, CONCLUSIONS, SUMMARY, AND IMPLICATIONS FOR FUTURE RESEARCH

This study was designed to investigate the possibility of sensory changes as a consequence of primary motor disease. Polio was selected as a primary motor pathology, tested within the framework of sensorimotor integration. Two functions mediated by sensory Ia nerve fibers were studied, joint position sense and modulation of muscle reflexes. Seven experimental hypotheses predicted directional changes of Ia mediated functions between the polio group and the non-polio group. This chapter presents the resolutions for the experimental hypotheses, discusses these findings within the context of sensorimotor integration, provides conclusions appropriate to these findings, and identifies areas of related future research.

Resolution of Hypotheses

Joint Position Sense Hypotheses

The first hypothesis predicts lower performance accuracy on the unperturbed knee joint flexion position matching task for the polio group in comparison to the non-polio group. The findings did not support the hypothesis. Two of the three dependent measures (CE and AE) revealed significant Group x Vibration Condition interactions. Therefore, it appears that for a non-perturbed motion both the polio and non-polio groups perform with equal accuracy.

The second joint position sense hypothesis predicts a difference in performance accuracy between groups when the knee joint positioning task is perturbed by vibration, such that the individuals who had polio would demonstrate less error than the non-polio group. This hypothesis is supported. The significant Group x Vibratory Condition interaction effects for CE and AE indicates that vibration powerfully influences the accuracy of those individuals who have not had polio, but had little effect on those participants who had polio.

Reflex Modulation Hypotheses

The first reflex modulation hypothesis, is supported, that in comparison with the non-polio group Hmaximum/Mmaximum ratios of the polio group are significantly reduced. Upholding this hypothesis shows that there is a significant difference between groups, such that the non-polio group demonstrates larger Hmaximum/Mmaximum ratios than the polio group.

The second reflex modulation hypothesis is not supported. The hypothesis predicted that individuals with polio would show a significant reduction in voluntary contraction facilitation as measured by percent facilitation above control values compared to non-polio individuals. Though there is a slight difference between the groups, with the non-polio group facilitating to a greater degree, this difference is not statistically significant.

The third and fourth reflex modulation hypotheses specify that individuals with polio would show significantly less inhibition than controls under the conditions of vibration and low frequency electrical stimulation respectively. Neither of these hypotheses is upheld.

The fifth hypothesis specific to the TVR indicating that individuals with polio would show a reduced TVR compared to the control group remains untested. This experiment provides neither support nor disagreement for this hypothesis.

Discussion

Sensorimotor integration provides the theoretical arena for investigating physiological and psychological mechanisms as they interact with input during motor actions (Brooks, 1986; MacKenzie & Marteniuk, 1985). It is readily accepted that there is a mutually dependent relationship between the sensory and motor systems (Abbs, Gracco, & Cole, 1984; Glencross, 1995; Marteniuk & MacKenzie, 1985). Researchers agree that sensorimotor integration is a fundamental aspect of motor control ranging from responses at the cellular level, such as altered motoneuron membrane excitability in response to Ia sensory stimulation, to overt behavioral responses such as human bimanual coordination (Gordon & Ghez, 1991; Marteniuk, MacKenzie, & Baba, 1984). The strength of sensorimotor linkages is also evidenced by changes in motor function as a result of sensory pathology as demonstrated in Ia sensory pathology eliminating the muscle stretch reflex or deafferentation leading to reduced bimanual coordination (Cope, Bonasera, & Nichols, 1994; Teasdale et al, 1994).

The Ia fibers originating from the muscle spindle provide an excellent physiologic model of sensorimotor integration (Boyd & Gladden, 1985; Gordon & Ghez, 1991). Therefore, the purpose of this dissertation is to test the prediction of Ia sensory system dysfunction in the presence of a primary motor disease poliomyelitis (Dalakas, Bartfeld, & Kurland, 1995; Halstead, 1995). Joint position sense and the muscle reflex modulation are

studied as representing functions mediated by Ia sensory fibers (Cordo, Gandevia, Hales, Burke, & Laird, 1993; Fisher, 1992; Matthews, 1990; Roll, Vedel, & Ribot, 1989).

Joint Position Sense

Several findings from the knee joint positioning experiment support the prediction of altered Ia function in individuals who have had polio. The most important finding is the significant Group x Vibratory interaction effect identified for the CE and AE dependent variables. This interaction effect shows that the polio group and non-polio group respond differently to vibration, with the non-polio group responding more strongly to vibratory perturbation than the polio group. The reason this finding is so crucial is that vibration has been consistently shown to induce altered accuracy in joint positioning as the result Ia sensory nerve fiber stimulation (Cordo, Gandevia, Hales, Burke, & Laird, 1993; Roll, Vedel, & Ribot, 1989). In the non-polio group, vibration produces a decrease in accuracy as evidenced by the marked increase in CE and AE, whereas only slight changes in CE and AE scores are found in the polio group during vibration.

These group differences to vibration are further highlighted by examining the direction (sign) of the mean CE scores. In motor control research, CE has been used traditionally as a measure of accuracy and it has both magnitude and direction components (Granit, 1972; Schmidt, 1988). Directionality is most important in regards to vibratory perturbation in that vibration of the lengthening tendon alters Ia firing rates, and consistently produces an undershooting of the target (Inglis & Frank, 1990; McCloskey, Cross, Honner, & Potter, 1983). This target undershooting indicates a negative score with a certain magnitude. Note that for this knee joint position experiment the mean CE score for the non-polio group is -3.7. However, the mean CE score for the polio group

with vibration is $.12^{\circ}$. This issue of directionality takes on even greater meaning when the individual CE responses within the groups are examined. The polio group shows that nine of 15 participants had a mean error in the positive direction, which means that they overshot the target. In contrast, only one of 15 non-polio participants reveal a mean error of overshooting.

While the major support for accepting the premise that there is a difference in Ia mediated joint position sense between the polio and non-polio group stems from the significant Group x Vibratory Condition interactions, several significant main effects are found. These vibratory condition main effects for the CE, ACE, and AE dependent measures indicate that the level of vibration used in this experiment is appropriate and generates the expected physiological response of altered joint position sense.

An additional important result from the knee joint positioning sense experiment is obtained by inspecting the CE, ACE and AE data for the groups under the no-vibration condition. For each dependent measure, the difference between groups is less than $.6^{\circ}$. This observation of equality between the groups is consistent with the literature in that magnitude differences of at least 1° are typically required before a significant difference is reported (Barrack, Skinner, Brunet, & Haddad, 1983; Perla, Frank, Fick, 1995; Skinner, Wyatt, Hodgdon, Conard, & Barrack, 1986). Furthermore, the no vibration condition CE values for both groups are quite small, and when compared to other studies, are in agreement with normal error magnitudes (Martin & Jessell, 1991; Skinner, Barrack, & Cook, 1981).

This equality between groups in a non-perturbed joint positioning task does in part help to explain the relatively high level of preserved function in individuals who have had

polio and their lack of obvious sensory deficits (Forster, 1978; Perry, Barnes, & Gronley, 1988; Windebank, 1995). The overall function of joint position sense is mediated by visual afferent, joint afferent, cutaneous afferent, and muscle spindle afferent systems (Lackner & Taublieb, 1984; Martin & Jessel, 1991; Paillard & Brouchon, 1974; Tardif-Gervet, Gilhodes, & Roll, 1986). Thus, joint position sense is a good example of how redundant systems contribute to the function of the whole (Abbs & Winstein, 1990, Winstein & Schmidt, 1989). In this experiment, visual feedback is unavailable, but the three remaining subsystems of joint, cutaneous, and muscle spindle afferents remain available. The presence of at least two of the redundant systems are required to maintain joint position sense (Kelso, Holt, & Flatt, 1980; Martin & Jessel, 1991). Indeed, in the polio group, even with a suspected deficit in the muscle afferent system, two subsystems (the cutaneous and joint afferent systems) remain intact providing accurate information about the tested positions.

These combined findings from the knee joint positioning experiments support the prediction of altered Ia function as a mediator of joint position sense in individuals who have had polio. The Group x Vibratory Condition interaction for both CE and AE provides strong evidence upholding the notion of altered Ia function; individuals who have had polio do not respond to vibratory perturbation.

H-Reflex Modulation

The H-reflex is the electrical analog to the physiological muscle stretch reflex (Fisher, 1992). Both are comprised of an afferent sensory limb mediated via the Ia sensory fibers, an efferent motor limb mediated via alpha motor neurons, and a mono and/or polysynaptic reflex arc located within the spinal cord (Burke, Gandevia, &

McKeon, 1984; Schieppati, 1987). Alterations of H-reflex function can occur as the result of changes in the Ia sensory fibers, alpha motor nerve fibers, alpha motor neurons, spinally mediated synaptic pathways, or descending supraspinal drive (Fisher, 1992, Kimura, 1989; Schieppati, Mardone, & Musazzi, 1986; Schieppati, 1987; Soliven & Maselli, 1992). Thus, changes in the H-reflex viewed in isolation are not a specific indicator of Ia pathology. The intent of the H-reflex modulation experiments was to demonstrate a pattern of altered H-reflex modulation, which when viewed together implicate the specific functions of Ia sensory afferents.

Hmaximum/Mmaximum ratios. The Hmaximum/Mmaximum ratio experiment clearly show a significant difference between groups, with the non-polio group demonstrating a 17% greater mean Hmaximum/Mmaximum ratio than the polio group. This difference is remarkable in the context of altered Ia function from several viewpoints. (1) all polio participants demonstrated a clean recordable voluntary EMG signal from the vastus medialis; (2) all polio participants demonstrated a recordable electrically evoked M-wave from the vastus medialis; (3) eight of the polio participants had good quadriceps strength with five having fair quadriceps strength; and (4) 10 of the 14 polio participants had no deep tendon reflex of the quadriceps muscle. When this information is combined with the results of the Hmaximum/Mmaximum ratio testing, dysfunction of the Ia system must be considered. In the presence of fair to good grade strength, why should there be an absence of the DTR? If there is a clean voluntary EMG signal and a recordable electrically evoked M-wave, why should there be no H-reflex or a diminished ratio of the H-reflex to M-wave amplitude? The possible answers to these questions, in view of an intact peripheral efferent reflex limb, suggest that either the afferent reflex limb (Ia

sensory fibers) or their spinal connections must be the site of dysfunction. It is particularly interesting to note that of the 14 polio participants five do not demonstrate an H-reflex, while in the non-polio group only one of 14 participants fails to demonstrate an H-reflex. This points to a remarkable difference between the groups, as over 1/3 of the polio participants show a complete failure of the H-reflex response.

The Hmaximum/Mmaximum ratio difference between the polio and non-polio groups is not an unexpected finding because others have reported differences in the H-reflex using techniques that differed from those used in this study (Pradkash, Sinha, Mulkherjee, Katiyar, & Dey, 1995; Soliven & Maselli, 1992). Also, one of the primary clinical diagnostic criteria for postpolio syndrome is that individuals demonstrate hyporeflexia or areflexia (Dalakas, 1995b). Then, the underlying puzzle is then to determine the reason for the diminished or absent muscle stretch reflexes. Electrically, Soliven and Maselli (1992) in a study of single motor unit H-reflexes found that those H-reflexes of the postpolio group were unstable and varied greatly in amplitude, causing them to suspect possible problems in the afferent limb of the reflex arc. The present findings are consistent with prior research and the predictions of sensorimotor integration. In the context of the integrating the physical and electrophysiological findings of individuals who have had polio, it appears that they show definite indications of altered Ia sensory nerve function.

A line of thinking that provides further support for rendering the conclusion of altered Ia sensory function as being at least partly responsible for the reduction in Hmaximum/Mmaximum ratios incorporates established neurophysiological findings with those of polio pathophysiology. The motoneuron pool for a given muscle is composed of

motor units of different sizes and levels of threshold excitability. The relationship between motoneuron size and excitability is that the smaller motoneurons have a lower threshold of excitability (Clamann, Gillies, Skinner, & Henneman, 1974; Henneman, Somjen, & Carpenter, 1965). The polio virus has been shown to have a greater affinity to infect and damage the larger motoneurons (Buchthal, 1949; Leestma, 1991). These larger motoneurons represent motoneurons with higher thresholds of excitability. Thus, in polio there is a preferential involvement of large motoneurons, resulting in a surviving population of motoneurons which is made up of a higher percentage of low threshold motoneurons compared to the non-pathological state. By deductive reasoning, a population made up of a higher percentage of low threshold motoneurons should show a greater Hmaximum/Mmaximum ratio than a population with a normal percentage of low threshold motoneurons. So without knowledge of clinical presentation one would predict that based on neurophysiology and the pathophysiology of polio that the Hmaximum/Mmaximum ratio would be larger for the polio group than for the non-polio group. Given that the present findings conflict with the prediction, evidence for Ia involvement as a factor contributing to the reduction of Hmaximum/Mmaximum ratios in the polio group is claimed.

Low frequency and vibratory inhibition. Previous researchers reported that Ia sensory fibers mediate the inhibitory effects of low frequency and vibratory stimulation. Both are thought to work at the presynaptic level of the Ia to alpha motor neuron synapse, though their mechanisms of action are different (Ashby, Stalberg, Winkler, & Hunter, 1987; Cook, 1968; Hultborn, Meunier, Morin, & Pierrot-Deseilligny, 1987; Ishikawa, Ott, Porter, & Stuart, 1966). These H-reflex modulation experiments were developed on the

premise that if the Ia system was not functioning normally, the polio group would show a decreased level of inhibition in response to low frequency electrical stimulation and vibratory stimulation. In regard to the lack of significant differences between the polio and non-polio groups, this premise was not supported. Several circumstances may have interacted in producing this result, including a lack of statistical power due to participant attrition and issues specific to experimental methodology. These factors are discussed more fully in the Limitations section.

Voluntary contraction facilitation. The H-reflex typically shows a marked facilitation in response to a preceding low level voluntary muscle contraction (Hultborn, Meunier, Pierrot-Deseilligny, & Shindo, 1986). This facilitation is in part a result of several inputs including stimulation of the Ia sensory fibers, an increase in excitability of the spinal interneurons, and an increase in the spinal neurons as a result of descending influences (Bongiovanni, Hagbarth, & Stjernberg, 1990; Nielsen, Sinkjaer, Toft, & Kagamihara, 1994; Schieppati, Nardone, & Musazzi, 1986). Thus, a rather strong facilitation was expected to occur in both groups with the supposition being that the polio group would show a lower level of facilitation as a result of the presumed deficit in Ia function. The present experiment only demonstrates some interesting trends between groups, although a main effect for contraction condition is statistically significant.

The distinct trend in the data approached the predicted direction for the non-polio group, demonstrating a greater facilitation of the H-reflex response than the polio group. Additionally, between the two groups, only members of the polio group fail to facilitate or even inhibit in response to voluntary contraction. This trend is further supported by prior research which has questioned the integrity of spinal connections and descending drive in

individuals who have had polio (Allen et al, 1994; Bodian, 1949; Soliven & Maselli, 1992). Specific to identifying the role of Ia muscle afferents, studies have shown the importance of intact muscle afferents in increasing motor drive and reducing the effect of presynaptic inhibition at the onset of muscle contraction (Burke, Cracies, Meunier, & Pierrot-Deseilligny, 1992; Macefield, Gandevia, Bigland-Ritchie, Gorman, & Burke, 1993). While the results from this experiment do not reveal a statistically significant Group x Condition interaction, the trend supports the contention of altered Ia system function.

Tonic vibration reflex. The tonic vibration reflex experiment, while providing an interesting opportunity to possibly differentiate monosynaptic versus polysynaptic components, did not succeed in producing the expected results. This experiment was aborted after 17 participants were tested because only two individuals, one in each group, demonstrated a tonic vibration reflex. This lack of response strongly indicates that the stimulation parameters used for this experiment are inadequate to generate the expected physiological response. In humans, vibration of a limb muscle produces two responses, inhibition of the monosynaptic reflex arc and the development of a reflex muscle contraction (Desmedt & Godaux, 1980). The inhibition of the monosynaptic reflex arc is the result of increasing presynaptic inhibition to homonymous Ia fibers, while the development of the reflex muscle contraction is mediated through complex polysynaptic pathways (Burke, Hagbarth, Wallin, & Lofstedt, 1980). The rationale for designing this experiment is based on it revealing a differential response when compared to the vibratory inhibition H-reflex experiment. A differential response such as there being no or reduced inhibition with vibration during the H-reflex study in the face of an intact tonic vibration reflex response would have led to the conclusion that monosynaptic Ia pathways are

more involved than the polysynaptic pathways. However, this experiment did not yield data on the expected reflex response.

Limitations

There are several possible limitations which may directly influence the results of the H-reflex experiments. For the purpose of discussion, they are grouped into two categories; statistical power and issues related to experimental methodology. The reduction of statistical power, because of participant attrition, dramatically alters the ability of the H-reflex inhibition and facilitation experiments to differentiate between the groups. In the inhibition experiments attrition of participants are the result of six pairs having one member without a recordable H-reflex, problems with baseline value instability, and an inability to tolerate the stimulation in the remaining excluded pairs. For the voluntary contraction facilitation experiment loss of participants is due to an inability to tolerate the stimulation as well as control value instability. Regardless of the reasons for participant attrition, there is a trend in the direction of hypothesized predictions for low frequency inhibition and voluntary contraction facilitation. The results may have proven statistically significant if a reasonable sample size was attained.

Methodological issues which may affect the outcomes include control values of the H-reflex for the inhibition experiments, pain associated with the femoral nerve electrical stimulation, and instability of the evoked responses in the polio group. A goal of using control value H-reflexes of 30%-50% of the maximum M-wave for the inhibition experiments was established based on the work of Crone et al.(1990). This criterion could not be achieved for many of the polio group participants. Additionally, the level of stimulation used in generating higher amplitude responses is uncomfortable, causing

participants to request that the stimulation be stopped or disrupting the stability of control baseline values. Thus, a lower level of stimulation may have effectively produced a floor effect limiting the amount of inhibition observed.

Based on participant comments and observing their physical reactions, pain associated with the electrical stimulation is identified as a cause of significant participant attrition. This is more pronounced in the experiments involving low frequency inhibition, as the frequencies of 1Hz and particularly 5Hz were poorly tolerated. This factor is also inconsistent across participants in that some are only moderately bothered, while others demonstrate strong adverse responses. These responses are demonstrable in the evoked potential recordings as greater levels of instability.

Concerning the level of stimulation, it is apparent that the polio group, as compared to the non-polio group, appeared to show a greater sensitivity to slight changes in stimulation strength as demonstrated by fluctuations in response values. This presents an interesting possibility concerning a mechanism related to polio pathology that may affect response magnitudes between the two groups. It is well-documented that polio leads to a loss of anterior horn cells and their motor units, and that the remaining motor units are of much greater size (Borg & Edstrom, 1995; Wiechers, 1988). Thus, the postpolio person has a smaller number of motor nerve fibers in their peripheral nerve, and each motor nerve fiber contributes a greater percentage to the total value. It follows that minor changes in the levels of electrical current flow can result in altered numbers of peripheral nerve fibers being stimulated. In the non-polio individual, with hundreds to thousands of peripheral motor nerve fibers, a slight reduction in the number of fibers depolarized will have little effect on the response amplitude. Conversely, a reduction in

the number of fibers depolarized in the polio individual may show a greater impact on response amplitude based on stimulus variability. This issue of response stability is of key importance for all of the experiments in which percent inhibition or percent facilitation of the H-reflex are examined, because of the greater influence any one motor nerve fiber has on total response in the individual with polio. Moreover, the response instability may have been a greater source of error.

Also specific to the polio group is a physiological response related to their disease pathophysiology that may contribute to response variability. A major point of pathology for the person who has had polio is the myoneural junction (Daube, Windebank, & Litchy, 1995; Maselli, Wollmann, & Roos, 1995). Failure of the myoneural junction in part or total can alter a motor unit's electrically evoked amplitude (Kimura, 1989). The use of the low frequency stimulation, particularly the 5Hz level, may have caused some aspect of myoneural junction failure which contributes to the inhibitory response though it is unrelated to the Ia sensory nerve fiber and alpha motor neuron synapse.

Lastly, it is surprising to note the lack of effect vibration had during the H-reflex experiment and the inability to generate a tonic vibration reflex in non-polio participants. Certainly this finding is not consistent with the literature (Burke, Hagbarth, Wallin, & Lofstedt, 1980; Desmedt & Godaux, 1980). The vibration amplitude in these experiments did not replicate the expected reflex responses, even though it is sufficient in producing the altered responses in the knee joint positioning task.

In spite of these potential limitations, these experiments provide a wide variety of findings that substantiate the idea that polio may indeed cause changes in those functions

mediated by the Ia sensory fibers. These findings are summarized in the following section, which addresses the conclusions of this project.

Conclusions

The conclusions for this series of experiments are specific to the question of Ia sensory nerve mediated functions in individuals who have had polio. This study was developed in response to the paradox presented between what is historically thought of as a primary motor disease without sensory loss and motor control theory which intimately relates sensory to motor and motor to sensory function (Bodian, 1949; Burtner & Woollacott, 1995; MacKenzie & Marteniuk, 1985; Perry, Barnes, & Gronley, 1988). Additionally, even though polio is thought to be without sensory consequences, the clinical findings of diminished stretch reflexes and frequent falling demand a clinician to challenge the assumption of normal sensory functions. Given the above as the setting for these experiments, the following conclusions are made.

(1). CE, ACE, and AE measures for a slow passive knee flexion joint positioning task without visual feedback and without vibratory perturbation are equal for groups of polio and non-polio participants. These data are in agreement with generally accepted physical findings for individuals who have had polio. Also, this conclusion is consistent with findings from other studies where proprioceptive inputs are experimentally or pathologically altered in single subsystems, leaving the other subsystems intact.

(2). CE and AE measures for a slow passive knee flexion joint positioning task without visual feedback show a markedly different response to vibratory perturbation between the polio and non-polio groups. This is a striking finding which can be taken as

clear support for a specific involvement of the Ia sensory fibers as a late consequence of polio.

(3). People who have polio have lower Hmaximum/Mmaximum ratios than those who have not had polio. This finding has been previously identified in polio studies involving single motor unit responses. This finding of lower Hmaximum/ Mmaximum ratios in those who have had polio supports the idea of altered Ia mediated sensory functions.

(4). Responses to H-reflex inhibition as the result of low frequency stimulation at 1Hz and 5Hz are not statistically different between the two groups. This finding of no difference is of questionable value in view of the loss of statistical power as the result of participant attrition.

(5). Vibratory inhibition of the H-reflex fails to show a difference between groups. Given the loss of statistical power because of participant attrition as well as the low control H-reflex values used in this experiment, this finding of no difference does not accurately contribute to the underlying question of Ia function in individuals who have had polio.

(6). The voluntary contraction facilitation experiment does not identify a difference between the polio and non-polio groups. Loss of statistical power also contributes to this finding. Though a strong trend was noted, demonstrating a reduction in the level of facilitation experienced by the polio group, application of these findings to a conclusion of Ia sensory function deficit is not warranted.

(7). In view of these experimental findings related to altered joint position sense and altered muscle reflex patterns in individuals who have had polio, good evidence exists

implicating involvement of the Ia sensory system. This involvement is in agreement with predictions generated under the theory of sensorimotor integration, that dysfunction of the motor system should lead to dysfunction in the sensory system.

Ramifications of Altered Ia Sensory Function

The ramifications of altered Ia function on issues specific to motor control span a wide range of possibilities. The Ia system plays a crucial role in variety of functions, ranging from cognitive awareness of limb position to a host of spinally mediated responses (Fredericks, 1996; Gordon & Ghez, 1991; Matthews, 1990). These functions and the potential for contributing to problems experienced by the post-polio person are discussed in the following paragraphs.

Specific to joint position sense and the broader category of proprioception, the Ia sensory system has been identified as being a contributing factor to the functional roles of coordination and balance (Brooks, 1986; Hulliger, 1993, Matthews, 1990). Specific to polio, a major problem and threat to the individuals health is falling. Reports of falling among members of polio support groups indicate that 30-60% experienced one or more falls in the last year (Kellogg, Cauraugh, Bauer, & Behrman, unpublished data). Furthermore, 80% or more of the individuals who are experiencing worsening symptoms related to polio report increasing weakness and fatigue as a primary complaint (Agre, 1995). The presence of a Ia deficit as identified in this study when coupled with increasing weakness certainly has the potential to contribute to the increased incidence of falls in these individuals. Additionally, 72% of the individuals presenting with increased symptoms reported difficulties with activities of daily living such as bathing, dressing, and job/homemaking skills (Agre, Rodriquez, & Sperling, 1989). If as suggested by Hulliger

(1993) the role of the Ia feedback is important for both short-term reflex modulation and maintenance of motor programs the identified deficit in Ia system functioning may be a significant contributing factor in the new difficulties being experienced in late polio.

The identification of fatigue as a primary concern in late polio has given rise to a list of potential sources. These include myoneural junction failure, local muscle metabolism differences, and altered function of the reticular activating system (Bruno, Frick, & Cohen, 1991; Grimby, Tollback, Muller, Larsson, 1996; Wiechers, 1988). Allen et al. (1994) have identified defective central and reflexive drive as contributing to weakness in polio. In normal muscle, Ia input has been shown to be an important source of facilitory stimulation to voluntarily activated anterior horn cells (Macefield, Gandevia, Bigland-Ritchie, Gorman, & Burke, 1993; Sanes & Everts, 1983). Given the role of the Ia system's contribution to assisting motor output, it is conceivable that a deficit in Ia function may contribute to this fatigue.

The Ia sensory system is a vital link as a source of muscle tone, the maintenance of upright posture, and movement coordination (Fredericks, 1996). Taking one issue at a time, the individual who has had polio can be shown to have subtle and not so subtle anomalies in each of these areas. As a result of the polio infection, muscle tone may progress from a spastic condition in the early acute phase to a flaccid condition in the later paralytic phase (Buchthal, 1949). In late polio, the form of paresis or paralysis is flaccid, with associated hypo or areflexia (Dalakas, 1995b). Specific to posture, individuals who have had polio demonstrate postural anomalies which may affect the spine, shoulder or pelvic girdles, and the extremities (Bennett, 1949; Smith & McDermott, 1987). And lastly, specific to movement coordination, persons with polio report increasing difficulty with

movement control in both activities of ambulation and daily living plus they are renowned for their unique movement substitution patterns (Perry, Barnes, Gronley, 1988; Smith & McDermott, 1987).

In summary, the Ia sensory system supports a wide variety of physiological functions which contribute to the efficient and coordinated use of voluntary muscle. Individuals who have had polio are experiencing increasing problems in the efficient and coordinated use of their muscles. Past research has indicated that while the possibility exists for sensory disturbances in polio, the clinical presentation of polio is generally free of obvious sensory disturbances (Bodian, 1949; Buchthal, 1949; Windebank, 1995). However, the results from this study have identified deficits in Ia function as it contributes to joint position sense and reflex modulation. These findings indicate that in addition to the well-recognized problems specific to the reorganization of the motor unit and myoneural junction, a deficit in Ia sensory mediated functions may be a contributing factor in the changes associated with the late effects of polio.

Clinical Implications

The results of this study indicate that Ia sensory mediated functions of joint position sense and reflex modulation are altered in response to polio. This finding has been suggested by prior researchers, but not clearly identified using a combination of psychophysical and neurophysiological experimental techniques (Bodian, 1949; Soliven & Maselli; 1992). Though not the focus of this study, a variety of potential clinical applications are presented here as future directions for research and clinical trial. Foremost of these is in the treatment of people with post-polio syndrome. These findings provide support for using treatment techniques directed at improving balance and

coordination. Currently, rehabilitation goals are most often specific for improving strength and lessening fatigue. However, with the primary deficit in post-polio syndrome being recognized as failure of the myoneural junction and a loss in motor units, these interventions have provided little benefit. By adding balance and coordination training, a shift in treatment emphasis may occur which focuses more on improving movement efficiency. This may prove to be more efficacious in improving function.

Another potential clinical application stems from using a combined experimental approach in identifying isolated sensory subsystem involvement. The use of vibratory perturbation in joint positioning task and muscle reflex modulation experiments collectively provide an intriguing avenue for the identification of Ia sensory deficits in other pathologies. The Ia sensory nerve fibers are frequently the first to become involved in both metabolic disease processes and in cases of nerve compression. Often in the earliest stages these conditions are difficult to diagnose. Perhaps adding the techniques used in this dissertation to the more traditional methods of clinical examination may potentially improve the early detection of these conditions.

Additional clinical applicability points arising from these findings include; (1) using polio as a model to study the reorganization of the spinal cord in response to motor neuron damage and death, (2) using vibration as a means to study potential reflex changes in response to acute and chronic joint injuries, and (3) using polio as a model of a primary motor disease within other theories of motor control to assist in the development of these theories as they can generalize to pathological conditions.

Summary

The findings and conclusions of these experiments strongly suggest that the functions of joint position sense and reflex modulation as mediated by the Ia sensory nerve fibers are altered in individuals who have had polio. The interaction results of the joint positioning experiment clearly provide evidence supporting the experimental predictions. When these results are combined with the significant differences between groups for the Hmaximum/Mmaximum H-reflex ratios and the data trends in the other H-reflex experiments, an overall interpretation of altered Ia sensory function as the result of polio is warranted.

Implications for Future Research

This study supports the sensorimotor integration prediction of altered sensory functions in response to a primary motor disease. Suggested areas of future research are listed below and cover areas specific to the topic of sensorimotor integration, polio, and other diseases.

(1). Using direct measurement methods, such as microneurography, to assess Ia sensory fiber function in individuals who have had polio. Microneurography involves measuring the nerve action potentials being carried by the peripheral Ia fibers. This quantification of these fibers may lead to conclusions concerning the number of fibers present, how well they conduct action potentials, and their sensitivity to specific levels of stretch. These variables would be directly measured from the Ia sensory nerve fibers, allowing very specific conclusions concerning Ia sensory fiber function.

(2). Conducting a continued line of research which focuses on muscle reflex modulation would be helpful in further exploring the ramifications of altered sensory input

in polio. Included in a series of experiments would be a repeat of the inhibition, facilitation, and TVR experiments as proposed in this dissertation. However, these manipulations should be further pilot tested to improve their experimental yield. In the case of the voluntary facilitation experiment, increasing the sample size and improving the control over contraction strength would improve the likelihood of finding significant group differences. For the TVR and vibratory inhibition experiments, increasing the strength of the vibratory stimulus and increasing the duration of vibration should be investigated.

Additional examples of studying reflex modulation would include an examination of reciprocal inhibition, heteronymous facilitation, and Ib mediation of muscle force modulation. The study of reciprocal inhibition and heteronymous facilitation are specific to the functions of Ia sensory fiber mediated functions and could be accomplished with techniques similar to those currently used. An examination of Ib mediation of force modulation would require the use of different experimental techniques which could include microneurographic studies or psychophysiologic studies designed to investigate single and multilimb interactions.

(3). Repeating the joint position sense and reflex modulation experiments on a group of individuals with a recent history of having had acute polio may assist in differentiating the Ia sensory changes as being the result of the acute disease or adaptive changes which occur over time. This is an exceptionally interesting idea, in that a comparison of acute pathology changes to chronic pathology changes will better elucidate the pathophysiology of polio. Furthermore, a comparative study of this sort will help to

develop the constructs of sensorimotor integration theory as it relates to issues of pathophysiology.

(4). An examination of polio from a theoretical perspective different from sensorimotor integration may be helpful in better understanding the adaptations occurring in polio that allow for relatively preserved function in the face of such severe motor deficits. In particular, viewing polio from a dynamical systems perspective, which emphasizes the emergent aspects of behavior without regard to isolated system function, would be most interesting. This is a particularly salient point in differentiating thought processes between clinical inquiry and laboratory research. Clinicians typically form their line of inquiry with a focus on, why is there system failure? I believe an equally important perspective can be attained from focusing on an alternative question, why does this system work? Viewing polio from a dynamical systems perspective may provide an impetus to change treatment paradigms from emphasizing what doesn't work, to maximizing the efficiency of those systems that do work.

(5). The application of the vibratory perturbation technique during a motion task has other clinical applicability. This technique may provide an early identification of pending Ia system failure in individuals at risk because of the underlying pathology such as diabetes or because of a hazardous work environments such as exposure to high levels of vibration or repetitious work at the extremes of joint motion. Diabetes is an example of a disease related to metabolic abnormalities in the use of blood sugar. It is a leading cause of peripheral polyneuropathy, which contributes to the disability and an increased incidence of amputation associated with this disease. Frequently the earliest sign of diabetic neuropathy is the reduction or absence of muscle stretch responses which are

mediated by the Ia sensory fibers. A similar scenario is noted for certain peripheral and spinal nerve entrapment syndromes. The larger sensory fibers, of which the Ia's are the largest, often demonstrate a greater sensitivity to compression syndromes. Next to prevention, early detection is a keystone in the appropriate medical management of these problems. The implication specific to the present findings is that the techniques used in determining Ia sensory system changes may prove helpful as diagnostic aides in the recognition of early Ia neuropathic conditions.

APPENDIX A

SAMPLE SIZE CALCULATIONS FOR JOINT POSITIONING TASK AND H-REFLEX EXPERIMENTS

Sample Size Calculations

Sample sizes were calculated based on techniques advocated from two separate sources (Marks, 1982; Shavelson, 1988).

Calculations were conducted with β set at .2 (power of .8) and α set at .05.

Concerning the joint positioning experiments a difference of 1° was chosen as being a meaningful difference, with σ being estimated from previous studies as 1.14.

$$\text{Using the formula } n = \frac{2(|z_\beta| + |z_\alpha|)^2}{\Delta^2}$$

$$\text{Where } \Delta = \frac{\text{Critical Difference}}{\sigma} \quad \Delta = 1/1.14 = .877$$

$$\text{For a one-tailed test: } \frac{2(.84 + 1.65)^2}{.877^2} = \frac{2(6.2)}{.769} = \frac{12.4}{.769} = 16$$

When sample size was determined from tables in the text by Marks (1982), Δ approximates .85, yielding a sample size of 9 for each group.

Sample size calculations were conducted for H-reflex experiments using the values from H-maximum to M-maximum ratios. Using 10% as a critical difference, and $\sigma = 8.7$ the calculated n using the formula results in:

$$\frac{2(.84 + 1.65)^2}{1.14^2} = \frac{12.4}{1.29} = 10$$

Sample size determined from table in the text by Marks (1982), Δ approximates 1.1, yielding a sample size of 6 in each group.

APPENDIX B
PARTICIPANT INTAKE FORMS

PARTICIPANT INTAKE FORM

Date: _____

Participant # _____

Name: _____

Age: _____

Gender: _____

SS#: _____

Mailing Address: _____

Phone#: _____

Check request submitted (date): _____

Check mailed (date): _____

POLIO HISTORY FORM

Participant# _____

Please answer the following questions. Where appropriate fill in the blank, circle the response, or complete the diagram.

1. Age _____

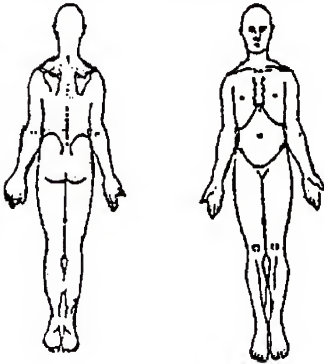
2. Gender Female Male

3. Race African American American Indian Asian
 Caucasian Spanish American Other

4. Age at which you had acute polio _____

5. Number of years required for rehabilitation _____

6. On the figure below please circle the body parts involved. Treat each arm/leg, the head and trunk as individual structures. Highlight the body part most severely involved.



7. What is your method of mobility?

Electric wheelchair/scooter
Walking with crutches
Unassisted walking

Manual wheelchair
Walking with canes

8. Do you wear leg orthotics/braces? _____

9. Do you use arm orthotics/braces? _____

10. What is your level of independence?

- Fully independent in all daily activities
- Need occasional assistance in daily activities
- Need frequent assistance in daily activities
- Require assistance in daily activities
- Fully dependent in some daily activities
- Fully dependent in all daily activities

11. Do you have difficulty climbing stairs? YES NO

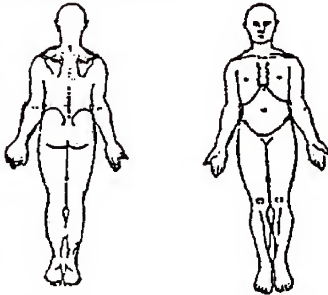
12. What activities do you require assistance with?

Transportation
Eating

Ambulation
Personal Care

Dressing

13. Do you have any areas of decreased sensation or numbness? _____. If yes please circle the body part involved.



14. Are you currently experiencing any changes in your capabilities? _____.

If yes please explain.

(Examples: Decreased endurance, muscle weakness, increase in falling, etc.)

15. What is your greatest concern in regards to your current postpolio status?

16. Are you currently being followed by a physician for your postpolio syndrome?

Yes _____ No _____

Name:

Phone number:

17. Are you currently being treated by a physician for any other medical condition?

Yes _____ No _____

If yes, please describe.

18. Are you taking any medications?

Yes _____ No _____

If yes, please list the medications?

19. Are you participating in any form of exercise training program?

Yes _____ No _____

If yes please give a brief description.

20. Is there any other issue that you would like to share concerning how postpolio syndrome is influencing your capabilities?

Participant# _____

NEUROMUSCULAR SCREENING FORM

Joint Range of Motion:

Hip: _____

Knee: _____

Ankle: _____

Muscle Strength:

Hip Flexors: _____

Knee Extensors: _____

Ankle Dorsiflexors: _____

Toe Extensors: _____

Hip Abductors: _____

Ankle Everters: _____

Hip Extensors: _____

Knee Flexors: _____

Ankle Plantar Flexors: _____

Toe Flexors: _____

Hip Adductors: _____

Ankle Inverters: _____

Sensation:

Light Touch

Saphenous Nerve _____

Peroneal Nerve _____

Deep _____

Superficial _____

Sural Nerve _____

Tibial Nerve _____

Tendon Tap Reflexes:

Knee Extensors: _____

Ankle Plantar Flexors: _____

APPENDIX C

INSTITUTIONAL REVIEW AND HUMAN SUBJECT CONSENT FORMS

University of Florida Institutional Review Board

1. TITLE OF PROJECT: Kinesthetic Loss in Post-polio Syndrome.

2. PRINCIPAL INVESTIGATOR(s):

Robert Kellogg, M.S., P.T., graduate student, Exercise & Sport Sciences Dept., 25 FLG, 392-9575.

James Cauraugh, Ph.D., Associate Professor, Exercise & Sport Sciences Dept., 25 FLG, 392-9575.

3. SUPERVISOR:

James Cauraugh, Ph.D., Associate Professor, Exercise & Sport Sciences Dept., 25 FLG, 392-9575.

4. DATES OF PROPOSED PROJECT:

20 November 1995 to 20 November 1996

5. SOURCE OF FUNDING FOR THE PROJECT:

Grant from the Foundation for Physical Therapy

6. SCIENTIFIC PURPOSE OF THE INVESTIGATION:

Individuals who have had polio provide an unusual model for the study of joint position sense, in particular the muscle spindle's contribution to joint position sense. As polio primarily effects motoric elements of the nervous system, it provides us with an opportunity to investigate the role of altered motor function on sensory processes. The specific purpose of this study is to measure the level of proprioceptive function in individuals with postpolio syndrome. Additional goals are: 1) to quantify the relationship between electrically evoked H-reflexes and electrically evoked distal motor compound muscle action potentials in individuals with postpolio syndrome and 2) to assess the effect of vibratory perturbation upon proprioception and reflex modulation in individuals with postpolio syndrome.

7. DESCRIBE THE RESEARCH METHODOLOGY IN NON-TECHNICAL LANGUAGE:

Twenty adult participants with postpolio syndrome and 20 neurologically normal age and gender matched adult participants will be tested for kinesthetic (joint position sense) function in a single joint motion of the knee, with and without vibratory perturbation. Additionally, participants will be tested for electrically evoked H-reflex responses, and electrically evoked motor responses. Dependent measures will include joint positioning accuracy, positioning error with vibration, and evoked response amplitudes of the H-reflex and motor evoked response.

Participants to be included in this study will be tested by manual muscle testing and must have between trace and fair strength of the calf and thigh muscle complexes. They must also have at least 90 degrees of passive knee motion, and have normal sensation to touch

Testing of the H-reflex and motor evoked potentials will require that the participants be positioned supine upon a padded table with the trunk and limbs well supported. The head will be maintained in a neutral position, the arms in a comfortable position, the knees flexed to 20 degrees, and the ankles in 0 degrees of dorsiflexion. Surface electrodes with an onsite preamplifier will be attached to the skin over the medial soleus and medial quadriceps muscles. A ground electrode will be placed over the proximal anterior tibia. Stimulating electrodes will be positioned over the tibial nerve in the popliteal fossa and the femoral nerve along the upper anterior thigh. The initial electrical responses will be recorded using progressively higher amounts of stimulation until a maximal amplitude muscle evoked response is achieved. This amplitude value will be recorded for later comparisons. The H-reflex will then be recorded using a stimulation strong to cause a response varying from 20-50% that of maximal. Four testing paradigms will be conducted: 1) H-reflex to motor maximum; 2) H-reflexes during tendon vibration; 3) H-reflexes following heteronymous nerve stimulation; and 4) H-reflexes triggered from a twitch contraction. Twenty responses will be recorded during each paradigm, for a total of 80 response, and stored for later analysis.

Kinesthesia will be tested with the participant sitting on a padded chair, with the knee and foot free to move. The lower leg will be attached a dynamometer for the purpose of movement speed control (2-5 degrees/second). Knee range of motion will be measured using an electrogoniometer taped to the knee. Participants will be passively moved through a 30 degree arc of motion while their eyes are closed. At predetermined positions the movement will be held stationary and the participant will be given three seconds to commit the position to memory. The knee will then be passively moved to the extended starting position. As the knee is allowed to passively flex the participant is to signal with a buzzer when their knee arrives at the memorized position. Testing will be conducted with and without vibratory perturbation. During the vibratory perturbation a vibrator will be positioned over the quadriceps tendon during the retest condition. Participants will complete 10 trials in both the no vibration and vibratory condition, they will have a 30 second rest between trials. Movement accuracy will be recorded by computer and stored for later analysis. Throughout the joint position testing surface EMG will be collected from the lateral quadriceps muscle.

All testing will be conducted on the same day. Participants will be given frequent rests to prevent fatigue, and refreshments will be provided. Total testing time should be less than three hours, with no one portion requiring more than 45 minutes.

8. POTENTIAL BENEFITS AND ANTICIPATED RISK:

No immediate benefits to the participants are foreseeable. However, the project may yield information important in the future treatment of individuals with postpolio syndrome. There are minimal psychological or physiological risk to the participant which include the possibility of fatigue and discomfort with the electrical stimulation. The level of discomfort associated with the electrical stimulation ranges from mild to moderate and can be described as a quick light to medium intensity pinch. Exclusion criteria involve peripheral nerve disease (they would be unable to respond to the stimulus), people with implanted electronic devices (pacemakers, hearing aids, etc.), individuals with artificial knee joints or hip joints, and pregnant women. Participants will be verbally screened prior to participation in this study for these criteria.

9. DESCRIBE HOW SUBJECTS WILL BE RECRUITED, THE NUMBER AND AGE OF THE SUBJECT, AND PROPOSED MONETARY COMPENSATION (IF ANY):

The 20 individuals with postpolio syndrome will be recruited from postpolio support groups throughout Florida. The 20 non postpolio individuals will be recruited through local health, fitness and social facilities. Participant ages are expected to range from 45 to 80 years. Each individual will be paid \$20.00 for their participation.

10. DESCRIBE THE INFORMED CONSENT PROCESS. INCLUDE A COPY OF THE INFORMED CONSENT DOCUMENT (if applicable).

After preliminary explanations and instructions, participants will receive the attached consent form to read and sign. Ample opportunity will be given for the participants to ask questions.

Principal Investigator

Supervisor

University of Florida
Motor Behavior Laboratory

Consent Form

- I am being asked to fill out a screening form which will provide the experimenter with information about my pattern of muscle weakness and medical history. I have been assured that all information is confidential to the extent provided by law, will be kept in a locked cabinet, and that my name will not be attached to the screening form.
- I will be tested for the muscle strength in my legs by manual muscle testing. I have been told that this testing may cause mild muscle soreness the following day.
- I will have the sensation of my legs tested by being touched with a cotton swab and a device with two prongs.
- I am being asked to participate in research in which I will be asked to move my knee to specific target points at a speed controlled by an exercise device. These motions will be done with and without visual feedback.
- I am being asked to move my knee to specific target points with the application of vibration to my knee tendon. The vibration may feel like a low grade tickle, and it may feel like it causes a gentle muscle contraction.
- I will be exposed to low grade electrical shocks applied to the back of my knee and the front of my thigh. This form of stimulation will cause a quick twitch type muscle contraction in my lower leg and thigh muscles. These contractions will be recorded with an oscilloscope and analyzed by a computer.
- I will be exposed to low amplitude vibration during the reflex testing, this may feel like a low grade tickle.
- I will be asked to gently but quickly contract my thigh muscle. This contraction will be associated with a low grade electrical shock.
- I have been informed that the electrical stimulation may be mildly to moderately uncomfortable. The degree of discomfort has been compared to a rapid light or moderate pinch.
- I have been informed that the testing procedure may cause a sense of fatigue.
- I have been told that the total testing time should be less than three hours, and will be accomplished on one day.
- It has been explained to me that these testing techniques are commonly used, and that there are no more than minimal risks associated with my participation.
- It has been explained to me that my responses in this study will be confidential to the extent provided by law, that my name will be removed from collected data records, and that these records will be locked in a cabinet in the Motor Behavior laboratory.
- I will receive \$20.00 for my participation in this study.
- I have been offered an answer to all inquiries about the experimental procedures. I have been told that any additional questions about this research project may be directed to Robert Kellogg, Department of Exercise and Sport Science, University of Florida, (352) 392-9575.
- I have been informed that any questions or concerns about my rights as a research participant can be directed to the UFIRB office, PO Box 112250, University of Florida, Gainesville, FL, 32611-2250.
- I have been told that I am free to withdraw my consent and to discontinue participation in the study at any time.
- I have been provided the following statement concerning liability of the University of Florida, the Board of Regents, and the State of Florida. "If I am injured during this study, as a result of the negligence of the principal investigator, the University of Florida, The Board of Regents of the State of Florida, and the State of Florida shall be liable only as provided by law. I may seek appropriate compensation for injury by contacting the Insurance Coordinator at 316 Stadium, University of Florida, (352) 392-2556."

-- I have read the statements and procedures described above.

I voluntarily agree to participate in this study and I have received a copy of this description.

Date

Signature of Participant

Signature of Experimenter

Please note that this project has been approved by the Institutional Review Board through December 7, 1996.

UNIVERSITY OF FLORIDA
INSTITUTIONAL REVIEW BOARD
PO Box 112250 / 114 PSY
Gainesville, FL 32611-2250

Harriet S. Hayes

phone: (904)-392-0433
fax: (904) 392-0433
e-mail: hayes@psych.ufl.edu

May 28, 1996

TO: Mr. Robert Kellogg and Dr. James Cauraugh
25 FLG

FROM: Harriet S. Hayes, Executive Secretary 
University of Florida Institutional
Review Board

SUBJECT: UFIRB Project #95.393
Kinesthetic Loss in Post-polio Syndrome

Your revised informed consent form (with additional changes highlighted) has been approved by the Chair of the University of Florida Institutional Review Board. Enclosed is the dated, IRB-approved informed consent to be used when recruiting participants for this research.

You are reminded approval of this project runs through December 7, 1996. If you have not completed the project by that time, please contact this office for instructions on how to obtain a renewal.

Should additional revisions of this project be necessary at a future date, please contact the UFIRB office prior to implementation for approval.

/h2

APPENDIX D

EXPERIMENTAL PROCEDURES, GUIDELINES AND CHECKLIST

PARTICIPANT ORDER ASSIGNMENT

ID#	Position Sense	H-reflex
01PP	NV	F LF V
02PP	VN	LF V F
03PP	NV	F LF V
04PP	VN	LF V F
05PP	NV	V LF F
06PP	VN	V LF F
07PP	NV	LF V F
08PP	VN	V LF F
09PP	NV	V F LF
10PP	VN	F V LF
11PP	NV	LF V F
12PP	VN	V FL F
13PP	NV	F V LF
14PP	VN	V F LF
15PP	NV	LF F V
01NP	NV	F LF V
02NP	VN	LF V F
03NP	NV	F LF V
04NP	VN	LF V F
05NP	NV	V LF F
06NP	VN	V LF F
07NP	NV	LF V F
08NP	VN	V LF F
09NP	NV	V F LF
10NP	VN	F V LF
11NP	NV	LF V F
12NP	VN	V LF F
13NP	NV	F V LF
14NP	VN	V F LF
15NP	NV	LF F V

PP = Postpolio
NP = No polio
N = No vibration
V = Vibration
F = Voluntary facilitation
LF = Low frequency depression

TESTING PATTERN FOR JOINT POSITION SENSE

<u>Joint Position</u>	<u>Voltage</u>	<u>Time Factor</u>
37	.79	5
44	1.0	9
54	1.3	7
32	.64	6
50	1.18	9
33	.67	6
53	1.27	8
42	.94	6
55	1.33	9
52	1.24	10

Joint position and time factor generated randomly

Voltage calculated by formula $[(X-30) \times .03] + .58$, where X is the randomly generated value between 30 and 60.

These values were used for all participants however, for each participant pair a different randomly generated order of trial presentations were used.

EXPERIMENTAL PROCEDURE CHECKOFF SHEET

1. Informed consent completed _____
2. Intake sheet completed _____
3. Medical history questionnaire completed _____
4. Neuromusculoskeletal screen completed _____
5. Joint position test check list
 - Cybox on _____
 - Speed set to 3°/second _____
 - Goniometer secured to knee _____
 - Computer and amplifiers on _____
 - Goniometer plugged into preamp. _____
 - Preamp. plugged into channel 1 A/D box _____
 - Buzzer plugged into channel 2 A/D box _____
 - Buzzer given to participant _____
 - Skin over vastus medialis cleaned and prepared _____
 - EMG electrode placed over vastus medialis _____
 - Ground electrode over anterior tibia _____
 - EMG electrode plugged into preamp. #1 _____
 - Preamp. cable to oscilloscope _____
 - Preamp. cable to channel 3 A/D box _____
 - Preamp. set to raw and 20Hz low _____
 - Ground electrode plugged into preamp. #1 _____
 - Check EMG signal on oscilloscope _____
 - Turn all power output to 0 on power amplifier _____
 - Plug in power amplifier for vibrator _____
 - Connect power amplifier to vibrator _____
 - Plug in LVDT _____
 - Connect LVDT to oscilloscope _____
 - Position vibrator over proximal patellar tendon _____
 - Adjust vibrator and power amplifier for
maximal force, with a .8mm displacement _____
 - Data acquisition settings
 - File name (# PP/NP N/V) _____
 - Channels 3 _____
 - Titles (ROM, Marker, EMG) _____
 - External Start/Stop (none) _____
 - Sample period (5ms) _____
 - Session duration (1200s) _____
 - Provide 5 practice trials
 - Position limb _____
 - Hold three seconds _____
 - Move limb to start position _____
 - Allow limb to flex _____

Time one minute then new trial	_____
Start computer	_____
Start test	_____
Repeat 10 times	_____
Follow ROM assignment sheet	_____
Repeat procedure for second condition	_____
Observe oscilloscope for any EMG activity	_____
Observe oscilloscope for LVDT reading stability	_____
Save each file	_____
Start new file with new file name	_____

6. M-wave and H-reflex maximum procedure check list

Computer acquisition settings	
File name (# PP/NP Max)	_____
Channels 1	_____
Titles (vastus medialis)	_____
External Start/Stop (none)	_____
Sample period (50 μ s)	_____
Session duration (1152s)	_____

Prepare skin over the vastus medialis, femoral triangle, and gluteal fold with light abrasion and isopropyl alcohol	_____
Apply conductive gel to EMG and stimulating electrodes (Yellow)	_____
Secure EMG electrode over vastus medialis	_____
Connect EMG to preamp #1	_____
Preamp set on raw, 1K, and 20Hz low	_____
Connect preamp to channel 1 A/D board	_____
Connect preamp to channel 1 oscilloscope	_____
Test for EMG signal quality	_____
Stimulator settings	
Turn stimulator on	_____
Turn S2 on	_____
Trigger cable from S2	_____
Trigger to external input	_____

M-wave and H-reflex maximum continued:	
oscilloscope	_____
Intensity to 0*****	_____
Stimulus duration 1ms	_____
Repeat rate 1/10s	_____
Delay 0	_____
S2 set off	_____
Connect leads to stimulus isolation unit (Yellow)	_____
Stimulus isolation unit (off)	_____
Place anode over gluteal fold	_____

Place cathode over femoral nerve	_____
Turn stimulus isolation unit on	_____
Slowly adjust stimulus intensity	_____
until getting an observable response	_____
on the oscilloscope	_____
Move cathode to best position	_____
Secure with foam pad and pelvic	_____
strap	_____
Return amplitude to 0	_____
Start computer	_____
Start test	_____
Set S2 to repeat	_____
Turn stimulus isolation unit on	_____
Increase stimulus intensity by	_____
5 V after every two stimuli	_____
Adjust stimulus by finer degrees	_____
as needed to determine H-max	_____
Progress the stimulus intensity	_____
5 V until M-max is achieved	_____

7. H-reflex voluntary facilitation procedure checklist

Computer acquisition settings	
File name (# PP/NP F)	_____
Channels 1	_____
Titles (vastus medialis)	_____
External Start/Stop (none)	_____
Sample period (50 μ s)	_____
Session duration (1152s)	_____
Connect EMG to preamp #1	_____
Preamp set on raw, 1K, and 20Hz low	_____
Connect t-split to BNC on preamp	_____
Connect one lead to channel 1 A/D board	_____
Voluntary facilitation continued:	
Connect other lead to pulse marker unit	_____
Connect one lead to channel 1 oscilloscope	_____
Test for EMG signal quality	_____
Instruct participant in generating a barely	_____
palpable vastus medialis contraction	_____
Adjust pulse unit marker sensitivity so	_____
that it is triggered by the low level	_____
contraction	_____
Connect pulse marker unit to single	_____
pass only switch unit	_____
Stimulator settings	
Turn stimulator on	_____
Turn S2 on	_____
Trigger cable from S2	_____
Trigger to external input	_____
oscilloscope	_____

Intensity to 15	_____
Stimulus duration 1ms	_____
Repeat rate 1/10s	_____
Delay 0	_____
S2 set off	_____
Connect stimulus isolation unit	_____
(Yellow) to constant current unit	_____
(Yellow)	_____
Stimulus isolation unit (off)	_____
Constant current unit (off)	_____
Constant current unit set to 0	_____
Turn stimulus isolation unit on	_____
Turn constant current unit on	_____
Slowly adjust stimulus intensity	_____
until control H-reflex is approximately	_____
10% of M-wave Max.	_____
Start computer	_____
Start test	_____
Record 10 control H-reflexes	_____
at rest	_____
Stop test	_____
Plug one pass switch unit into	_____
S2 trigger input	_____
Set S2 on external	_____
Restart computer	_____
Repeat 10 trials, coaching the	_____
participant to generate a low grade	_____
contraction 1/10s	_____
Stop test and save	_____

8. Low frequency stimulation procedure checklist

Computer acquisition settings	
File name (# PP/NP LF)	_____
Channel 1	_____
Title (vastus medialis)	_____
External Start/Stop (none)	_____
Sample period (50 μ s)	_____
Session duration (1152s)	_____

Connect EMG from vastus medialis	
to preamp #1	_____
Preamp set on raw, 1K, and 20Hz low	_____
Connect one lead to channel 1 A/D board	_____
Connect one lead to channel 1 oscilloscope	_____
Test for EMG signal quality	_____

Stimulator settings:

Turn stimulator on	_____
Turn S2 on	_____
Trigger cable from S2	_____

Intensity to 15	_____
Stimulus duration 1ms	_____
Delay 0	_____
Stimulus rate 1every 10 s.	_____
S2 set off	_____
Connect stimulus isolation unit	_____
(Yellow) to constant current unit	_____
(Yellow)	_____
Stimulus isolation unit (off)	_____
Constant current unit (off)	_____
Constant current unit set to 0	_____
Turn stimulus isolation unit on	_____
Turn constant current unit on	_____
Set S2 to repeat	_____
Slowly adjust stimulus intensity	_____
until control H-reflex is approximately	_____
20-30% of M-wave Max.	_____
Start computer	_____
Start test	_____
Record 10 control H-reflexes	_____
at rest	_____
Stop test	_____
Restart computer	_____
Repeat 10 trials with stimulation	_____
frequency 1Hz	_____
Stop test and save	_____
Turn constant current unit off, adjust	_____
to 0 output	_____
Set S2 off	_____
Turn power amplifier output to 0	_____
and turn off	_____

9. Vibratory inhibition procedure checklist:

Computer acquisition settings	
File name (# PP/NP V)	_____
Channels 1	_____
Titles (vastus medialis)	_____
External Start/Stop (none)	_____
Sample period (50 μ s)	_____
Session duration (1152s)	_____
Output file:	
# of channels 3	_____
# of trials 1000 (10.0s)	_____
base address 800	_____
sample period 999 μ s	_____
scale range +/- 10 V	_____
Connect EMG to preamp #1	_____
Preamp set on raw, 1K, and 20Hz low	_____

- Connect one lead to channel 1 A/D board _____
- Connect one lead to channel 1 oscilloscope _____
- Test for EMG signal quality _____
- Connect signal output cable channel 1 to power amplifier _____
- Connect signal output cable channel 2 to grass trigger _____
- Connect signal output cable channel 3 to computer trigger _____
- Turn all power output to 0 on power amplifier _____
- Plug in power amplifier for vibrator _____
- Connect power amplifier to vibrator _____
- Plug in LVDT _____
- Connect LVDT to oscilloscope _____
- Position vibrator over proximal patellar tendon _____
- Adjust vibrator and power amplifier for maximal force, with a .5mm displacement _____
- Stimulator settings:
 - Turn stimulator on _____
 - Turn S2 on _____
 - Trigger cable from S2 _____
 - Intensity to 15 _____
 - Stimulus duration 1ms _____
 - External trigger S2 _____
 - Delay 0 _____
 - S2 set off _____
 - Connect stimulus isolation unit (Yellow) to constant current unit (Yellow) _____
 - Stimulus isolation unit (off) _____
 - Constant current unit (off) _____
 - Constant current unit set to 0 _____
 - Turn stimulus isolation unit on _____
 - Turn constant current unit on _____
 - Set S2 to repeat _____
 - Slowly adjust stimulus intensity until control H-reflex is approximately 20-30% of M-wave Max. _____
 - Start computer _____
 - Start test _____
 - Record 10 control H-reflexes at rest _____
 - Stop test _____
 - Enable computer control of output S2 trigger input _____
 - Set S2 on repeat _____
 - Restart computer _____
 - Repeat 10 trials with vibration _____
 - Stop test and save _____
 - Turn constant current unit off, adjust to 0 output _____

Set S2 off _____
Turn power amplifier output to 0 _____
and turn off _____

APPENDIX E

PRELIMINARY STUDIES AND PILOT WORK

Instrumentation Pilot Work #1 Electrogoniometry Linearity

The purpose of this pilot work was to determine the sensitivity and linearity of the electrogoniometer. This was accomplished by using oscilloscope to measure the electrical voltage generated by the electrogoniometer across a preselected series of positions from 9° to 78° . These positions were repeated three times on two different days. Figure 16 and 17 are graphs of the linear relationship between degrees and volts measured through different system components.

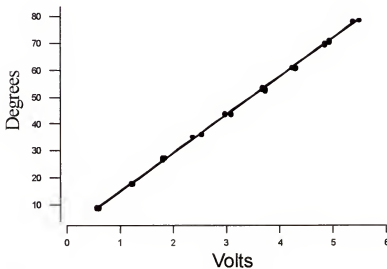


Figure 16. Linear relationship between volts and degrees using oscilloscope and electrogoniometer.

Pearson Product correlation of volts to degrees = 1
R squared = .99

Regression equation for degrees to volts is: $\text{Volts} = -.0410 + .0699X_1$

Linearity of Electrogoniometer as Measured with Computer

The purpose of this pilot was to determine the linearity of electrogoniometric measurements through the entire system of transducer, amplifier, computer and my measurement. Figure 17 illustrates the linearity of this measurement.

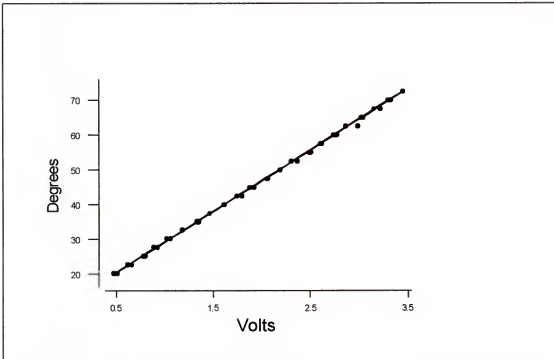


Figure 17. Linearity of volts to degrees measured through system to computer.

Pearson product correlation = 1

R-squared = .99

Instrumentation Pilot Work #3
Linearity of Distance Transducer (LVDT)

The purpose of this pilot work was to determine the linearity of voltage readings to measures of distance (mm) using the LVDT sensor as measured from the oscilloscope. Figure 18 displays this relationship. This measure is important in determining the amplitude of vibration, which will be adjusted and observed online during experiments requiring vibration.

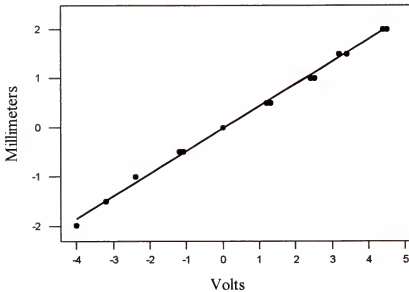


Figure 18. Linear relationship between volts and extent of pushrod movement.

Pearson product correlation = .99

R-squared = .99

Intraclass Correlation Coefficient Calculations
for ROM, vibrator excursion, and evoked potential latencies

Intraclass correlation coefficients (ICCs) were calculated for my ability to repeat measures for electrogoniometric ROM, linear excursions of the vibrator push rod through the linear velocity detector, and takeoff latencies of the H-reflex. Measures were taken on three separate occasions. The ICC was calculated by using the form ICC (3, 1) as described in Portney and Watkins (1993), and recommended for testing intrarater reliability.

ICC for electrogoniometric measurements = 1

ICC for linear excursions of the vibrator push rod = .88

ICC for takeoff latency of the H-reflex = .98

Preliminary Pilot Study #1: Joint Position Sense

This set of testing was conducted to refine the procedures to be used in the knee joint positioning experiments. Equipment used included the Cybex II dynamometer, electrogoniometer, amplifier bank, computer, and therapeutic hand held vibrator.

Adopting an experimental paradigm developed by Skinner, Barrack, and Cook (1982) the knee will be passively flexed at a constant velocity to a predetermined position. The participant will be given 3 s to remember the position. The knee will then be passively extended, then allowed to passively flex. The participant is to ring a buzzer when the knee reaches the memorized position. The rationale for adopting this procedure is: 1) to avoid fatigue; 2) the contralateral limb in individuals who have had polio may not be “normal”, thus a paired limb position matching task may not be appropriate; and 3) the use of a visual scale would add an additional level of comprehension difficulty.

Three individuals were tested. Mean joint positioning constant error ranged from 1° to 7°s. All individuals tolerated testing well, without complaints of discomfort. Additionally a hand held vibrator was applied to test the task for identifying the level of vibratory perturbation undershoot. Constant error ranged from 7° to 15°s. Two participants indicated that a longer waiting period may be necessary between vibration trials, as they felt that they did not recover from the vibratory sensation of the preceding trial in 15s. All participants reported using different cognitive strategies in assisting them in the accomplishment of the task. One participant was tested at different speeds of 3, 4, 5, and 8°s per second. Constant error values between the three speeds was not significantly different.

Conclusions: Values determined from these participants corresponded well with values reported in the literature. Vibration of the quadriceps tendon during passive knee flexion caused a predictable undershoot, resulting in increased error scores. The use of a 3°/s angular velocity is acceptable, and may help in preventing confounding a faster

velocity with issues of coincident timing. The time interval between trials will be extended to 60s.

Preliminary Pilot Study #2: Quadriceps H-Reflex

The purpose of this pilot work on four individuals was to refine the technique for collecting quadriceps H-reflex data.

Participant #1: Tested in a semireclined position on the Cybex bench with recording leads over the vastus lateralis and vastus medialis. Stimulation was provided at both the femoral triangle and over Hunter's canal. Recordings were taken from both the vastus medialis and vastus lateralis muscles. Initial impressions were that in this subject the response was not obtainable, however, on subsequent waveform inspection a small amplitude H-reflex was present and tightly coupled with the tail of the M-wave.

Participant #2: Testing done in a fully reclined position, recording from the vastus medialis. Stimulation was provided over both the femoral triangle and over Hunter's canal. H-reflexes were obtained from both positions. The H-reflex from stimulation over Hunter's canal was more discrete from the M-wave, however, it was of smaller amplitude than the H-reflex from the femoral triangle.

Participant #3 & #4: Testing done in a fully reclined position, recording from the vastus medialis. Stimulation provided over the femoral triangle. Both participants demonstrated an easily recorded H-reflex.

Conclusions: Findings from these pilot studies indicate that testing is best accomplished in a fully supine position, recording from the vastus medialis, and stimulating over the femoral triangle. Figure 19 represents recorded quadriceps M-wave and H-reflex waveforms.

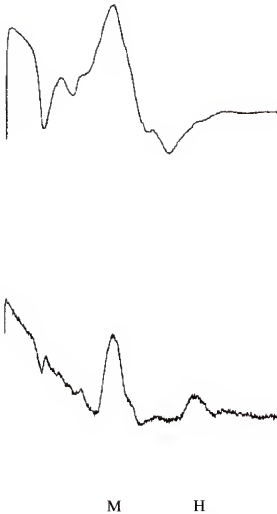


Figure 19. Evoked potential responses from the quadriceps muscle. The top tracing represents the maximal M-wave response. The bottom tracing represents the M-wave response (labeled by an M) and the H-reflex response (labeled by an H)

Preliminary Pilot Study #3: Homonymous Voluntary Facilitation

The purpose of this study was to develop and refine my technique in triggering the electrical stimulation from a low grade voluntary muscle contraction. This required the use of the signal marker generator and interfacing it with the Grass electrical stimulator.

Participant #1 was instructed to generate a low grade, quick twitch, isometric muscle contraction of his quadriceps. The amplified signal was fed into the pulse marker

generator, which has an adjustable sensitivity. With training the participant was able to generate a barely palpable contraction. When this contraction triggered the pulse marker unit, a markedly facilitated H-reflex resulted. On occasion the participant experienced a rapid train of electrical stimulations, causing a marked muscle contraction.

Participant #2 instructed as above. Results also showed a markedly facilitated H-reflex, and this participant also experienced the rapid train of electrical stimuli. It seems that a positive feed back loop is developing such that the voluntary contraction triggers an electrical stimulus, which triggers an H-reflex, which triggers an electrical stimulus, which triggers an H-reflex again, and the cycle repeats. This experience is uncomfortable as the contraction approaches tetany. The marker generator unit can only issue a pulse every 30 ms. This would result in approximately 30 impulses per second, which results in a fairly strong muscle contraction.

Participant #3 instructed as above. To prevent the repeated stimuli, I held the stimulator manual override switch in my hand. On seeing the pulse marker unit light turn on, I released the switch. Most often this prevented more than one shock from occurring. However, on several trials the participant received 2 or three stimuli before I could release the switch (a limitation of my reaction time). Continued to show a marked H-reflex facilitation.

Participant #4 & #5 same as in #3.

Conclusions: Findings from this set of pilot experiments indicate that I will need to have a single pass switch box constructed to prevent more than one marker pulse from triggering the stimulator. All subjects did show a marked facilitation of the H-reflex as expected. Figure 20 is illustrative of the waveforms recorded during the voluntary contraction facilitation experiment.

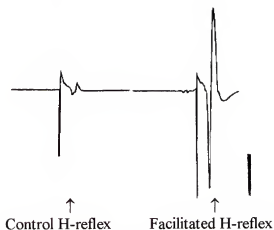


Figure 20. Demonstration of the effect of voluntary contraction on the amplitude of the H-reflex. The second waveform (labeled facilitated H-reflex) was recorded with a level of stimulation equal to that of the control H-reflex, but following a low grade voluntary muscle contraction.

Preliminary Pilot Study #4: Homonymous Vibratory Inhibition

The purpose of these experiments were to develop and refine my technique in the use of vibration as an inhibitory tool in H-reflex modulation.

Participant #1. The vibrator used in this study was a hand held therapeutic vibrator. This vibrator allowed minimal control over frequency, amplitude or waveform. Vibration was applied to the patellar tendon 1-5 seconds prior to the generation of an H-reflex. Inhibition of the H-reflex was mild to moderate in scope.

Participant #2. The hand held vibrator was again used. Attempted to vary the amount of pressure being placed on the tendon. Level of inhibition seemed to be related to amount of pressure.

Conclusions: The results of these experiments show that vibration does indeed cause an inhibitory effect as evidenced in Figure 21. However, the parameters of vibration must be more tightly controlled. Additional pilot work will be carried out upon the receipt of the Bruel & Kjaer vibrator which will be computer interfaced and driven.

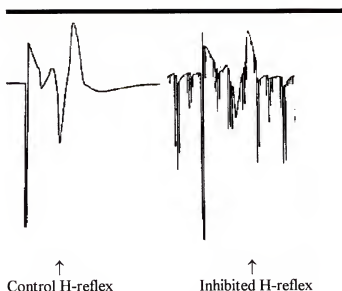


Figure 21. Inhibition of the H-reflex with vibratory stimulation. Also note that the therapeutic vibrator also caused significant interference in the baseline activity of the evoked response.

Preliminary Pilot Study # 5: Heteronymous Electrical Facilitation

The purpose of these experiences were to develop and refine my technique in the dual stimulation paradigm required for heteronymous facilitation.

Participant #1. Individual was instrumented for recordings from both the quadriceps and soleus muscles. The soleus muscle was stimulated via the tibial nerve in the popliteal fossa, and the quadriceps via the femoral nerve in the femoral triangle. The difficult task in this experiment is timing the two stimulations so that the stimulation mediated via the femoral nerve arrives at soleus motor neuron pool immediately prior to the tibial nerve stimulation. Recent work indicates that a delay of 5-6 ms is most commonly required for femoral to tibial nerve facilitation to be maximal. Gradually increasing the delay, maximal effect was noted between 4.8 and 5 ms.

Participant #2. Same as above. Delay was slightly longer with maximum effect noted at 5.5 ms.

Participant #3. Intent of this experiment was to further refine my technique by using a current probe to more finely tune the level of femoral nerve stimulation. By using

the current probe stimulation of the femoral nerve can be precisely adjusted to 1-1.1 x the motor threshold current level. Some fluctuation in the baseline current readings was noted. Participant demonstrated good facilitation.

Participant #4. Recognized that I have been determining the excitability curve in the wrong direction. Started protocol with a 10 ms delay, moving to shorter and shorter delays. Intuitively this procedure makes more sense. Will incorporate full recruitment curve into data collection sequence. This may prove to be an additional finding of difference between individuals with and without polio.

Conclusions: A very difficult technique, many variables requiring attention. Will include facilitation recruitment curves as part of the data collection. Process is time consuming, requiring 1-2 hours of testing for clean results. In spite of the difficulty, this technique provides an opportunity to evaluate the sensory contributions of femoral nerve Ia fibers to a different motor neuron pool. See Figure 22 for a display of the facilitatory effect between the femoral nerve and the tibial nerve evoked reflex.

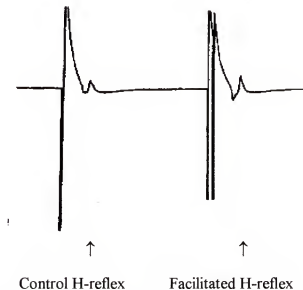


Figure 22. Facilitation of the tibial H-reflex with heteronymous stimulation of the femoral nerve.

Preliminary Pilot Study #6: Use of Bruel and Kjaer Vibrator

The purpose of this study was to incorporate the Bruel and Kjaer vibrator into the H-reflex experiments. In order to accomplish this the Datapac acquisition system was reconfigured so that the computer triggered and drove the vibrator prior to triggering the electrical stimulator. Additionally a third computer generated trigger was used to retrigger the entire computer system ever 10 s for 10 repetitions.

Participant #1. Tolerated testing well. Showed a mild depression of H-reflex response. Vibration was given at a frequency of 200Hz, for three pulses, occurring 55 ms prior to electrical stimulation.

Participant #2. Altered stimulation parameters such that vibration given at 100Hz, for 10 pulses, occurring so that the electrical stimulation is given on the 7th vibratory pulse. A greater level of H-reflex depression noted, with increasing the number of vibratory stimuli.

Conclusions: Have been able to interface the computer and vibrator. No electrical interference noted in EMG signal using the Bruel and Kjaer vibrator (marked interference noted when using the therapeutic vibrator). Will increase the train of vibratory impulses to 10, and deliver the electrical stimulation during the 7th stimuli.

Pilot Study Incorporating All Experiments in
Persons with Polio Compared to Age Matched Controls

The purpose of this study was to conduct complete pilot work on 3 individuals who have had polio and 3 control individuals for all experiments. The testing time required for each individual was approximately four hours. Descriptive statistics for each experiment follow. ROM data is expressed in volts, where .03 volts = 1°. H-reflex data is presented as percent change from control, with positive values indicating facilitation and negative values indicating inhibition.

Abbreviations:

NP = controls PP = individuals who have had polio

Vib = vibration

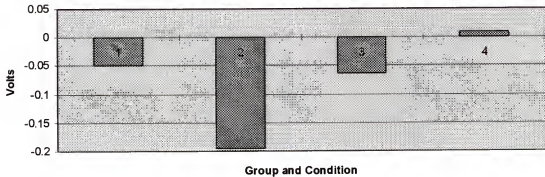
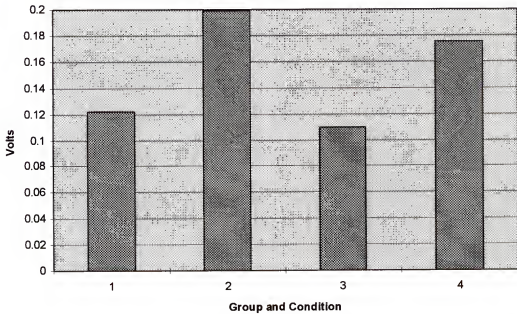


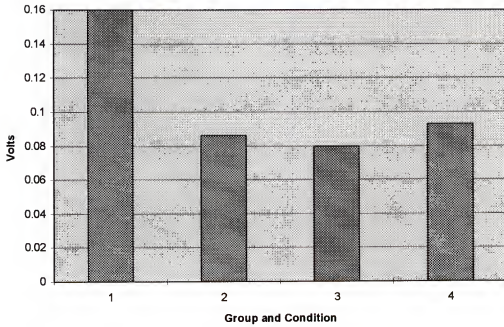
Figure 23. Graphic display, raw data, and mean group/condition values for CE data.

Positive values represent an overshoot of the target, negative values represent an undershoot of the target. Of a combined total of 120 trials, 42 were positive. During vibration only 2 trials were positive in persons who have not had polio, while in the polio group 12 responses were positive.



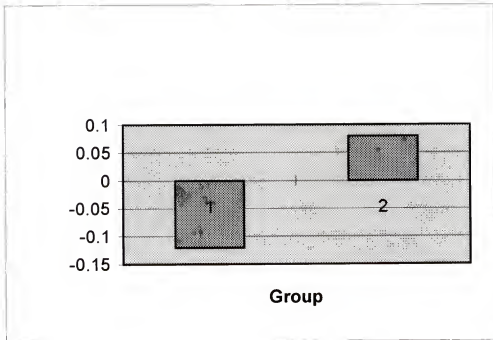
	NP No Vib	NP Vib	PP No Vib	PP Vib
	.076	.143	.051	.092
	.256	.215	.236	.146
	.036	.241	.072	.287
Mean	.122	.199	.11	.175
SD	.11	.05	.10	.10

Figure 24. Graphic display, raw data, and mean group/condition values for ACE data.



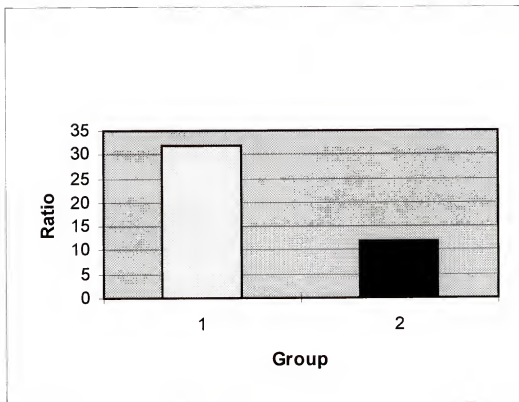
	NP No Vib	NP Vib	PP No Vib	PP Vib
	.051	.122	.063	.061
	.356	.128	.104	.090
	.074	.008	.074	.129
Mean	.160	.086	.080	.093
SD	.169	.060	.021	.034

Figure 25. Graphic display, raw data, and mean group/condition values for VE data.



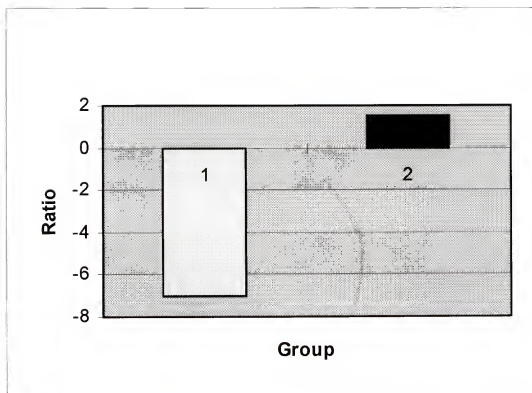
	NP	PP
	-.244	-.081
	-.019	.089
	-.10	.241
MEAN	-.12	.08
SD	.11	.16

Figure 26. Graphic display, raw data, and mean group/condition values for CE differences between conditions of vibration for each group from the joint positioning task experiment.



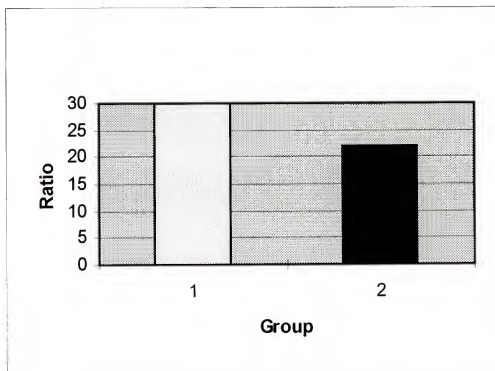
	NP	PP
	26%	4%
	42%	24%
	28%	8%
Mean	32%	12%
SD	8.7%	10.5%

Figure 27. Graphic display, raw data, and mean group values for Hmaximum/Mmaximum ratios.



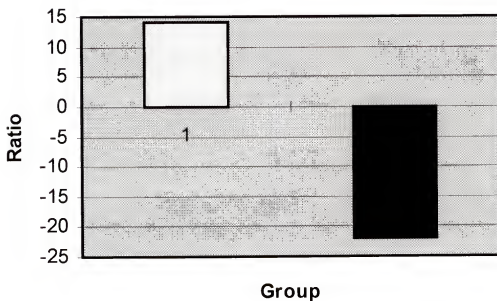
	NP	PP
	-5%	1%
	-4%	2%
	-12%	
Mean	-7%	1.5%
SD	4.3%	.5%

Figure 28. Graphic display, raw data, and mean group values for vibratory inhibition of the H-reflex.



	NP	PP
	20%	-1%
	23%	16%
	48 %	52%
Mean	30%	22%
SD	15%	27%

Figure 29. Graphic display, raw data, and mean group values for voluntary contraction facilitation of the H-reflex



	NP	PP
	30%	-22%
	10%	
	2%	
Mean	14%	
SD	14%	

Figure 30. Graphic display, raw data, and mean group values for heteronymous facilitation of the H-reflex.

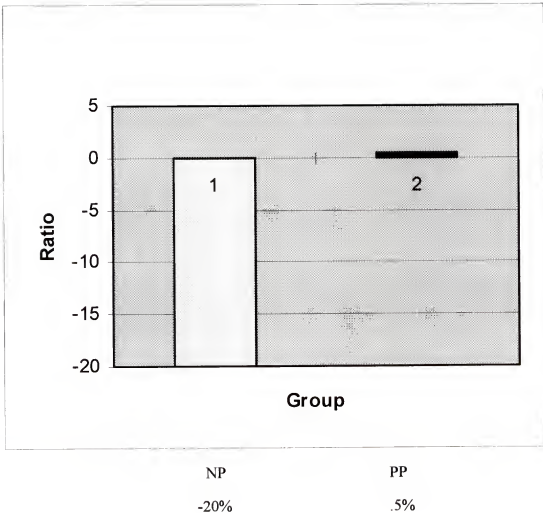


Figure 31. Graphic display, raw data values for low frequency inhibition of the H-reflex

Conclusions as a Result of the Preliminary Studies and Pilot Work

1. The electrogoniometer and LVDT are highly accurate measurement devices, with excellent linearity across the ranges to be measured in this study.
2. The investigator's use of the measurement tools being used in this study (electromyography, goniometry, and linear distance), show excellent reliability based on the calculated ICCs.
3. In all pilot participants tested a quadriceps H-reflex was obtainable. In two of the pilot participants, this response was of marginal quality and posed measurement difficulties

related to accurate latency and amplitude measures.

4. All participants who had polio generated a quadriceps H-reflex.
5. Using an angular velocity of $3^\circ/\text{s}$ resulted in error values consistent with those reported in the literature.
6. Increasing the intertrial interval to 60s decreased the carryover sensations during the vibration condition.
7. In an effort to increase participant comfort, and to control unwanted movement, a padded neck pillow and foam padded leg splint will be used during testing.
8. Testing as originally planned required 4-4.5 hours of work. The long testing time was a result of the experiment involving heteronymous facilitation (often requiring 1-1.5 hours). This experiment showed a poor yield of data from the individuals with polio as a result of concomitant soleus involvement, only 1 of 3 had a recordable response. Therefore, heteronymous facilitation will be dropped from the study.
9. In place of heteronymous facilitation, low frequency electrical inhibition will be substituted. In the one experimental pair serving as a pilot for this technique a large differential response was noted.
10. No untoward reactions were reported as a result of the joint positioning or H-reflex experimental procedures. The greatest complaint concerned the discomfort caused by resting in one position for several hours. This will be minimized by the removal of the heteronymous facilitation experiment, and by encouraging the participant to change positions during periods when data is not being collected.
11. In the process of analyzing the data from the H-reflex experiments I became concerned that the measures of amplitude based on .5 and 1 ms from the takeoff were of questionable use. Often the values were less than the takeoff value, in the presence of a positively rising slope. This leads me to believe that these measures lack the needed accuracy for this set of experiments. Therefore, I will only be using peak to peak measures of amplitude.
12. Based on sample size calculations from two sources, with $\alpha = .05$ and $\beta = .2$, 15 participants in each group should provide ample opportunity at achieving statistical significance.

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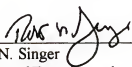
BIOGRAPHICAL SKETCH

Robert Michael Kellogg was born on June 21, 1955, in Oakland, California. He graduated from Paradise High School in 1973. He completed his undergraduate work at Chemeketa Community College in Salem, Oregon, and Southern Oregon State College in Ashland, Oregon, receiving a Bachelor of Science degree in general studies in 1978. He attended the Mayo Clinic's Program in Physical Therapy, graduating in 1980. In August of 1980 he accepted a commission into the United States Navy and has been on continuous active duty since. He has been assigned as a physical therapist to the Naval Hospitals Bethesda, San Diego, and Millington. In 1990 he earned a Master of Science degree from the University of Kentucky. He is currently assigned to the University of Florida, where he is a full time doctoral student in the College of Health and Human Performance, Department of Exercise and Sport Sciences.

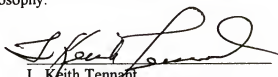
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James H. Cauraugh, Chair
Associate Professor of Exercise and
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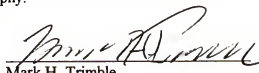
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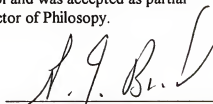


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This dissertation was submitted to the Graduate Faculty of the College of Health and Human Performance and to the Graduate School and was accepted as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

December, 1996



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